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ORIGINAL COMMUNICATIONS.

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CLINICAL REMARKS ON THE USE OF SURGICAL DIATHERMY FOR MALIGNANT TUMORS IN THE ANTERIOR AIR PASSAGES.*

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Few methods of treatment have impressed me so much as surgical diathermy. By means of diathermy it is possible to eradicate malignant neoplasms in the nose, pharynx and larynx without causing much inconvenience to the patient.

As treatment by diathermy has been known and practiced for a number of years, I don't suppose that what I am going to tell you will be a novelty to you, for in our literature of the last years you will find numerous papers giving a thorough description of this method; furthermore, I am convinced that many of my excellent American colleagues have had more experience in this matter than I, and I can in this respect refer to William Clark, of Philadelphia, who in 1918, in Vol. 71 of the *Jour. A. M. A.*, page 1365, published a paper, "Cancer of the Oral Cavity, Jaws and Throat", in which he analyzed the results of 200 cases, treated with electrocoagulation.

Clark writes as follows: "Electrothermic methods are peculiarly adapted to the treatment of cancer within the mouth. Malignant tissue, including bone, occurring in any part of the oral cavity, comprising the lips, buccal surface, tongue, floor of the mouth, alveolus and hard palate, antrum, tonsils, pharynx, epiglottis and proximal

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end of the esophagus, may be destroyed with one electrothermic operation."

My address must be considered as an expression of my private opinion of the advantage of diathermy in malignant tumors in nose, pharynx and larynx, illustrated by my personal experiences.

I will not discuss the use of diathermy in tuberculous affections of the mucous membrane, such as lupus, tubercular ulcerations and similar superficial and less serious affections, the treatment of which by galvanocaustic, diathermy, etc., presents no difficulties. I shall only give an account of my experiences with surgical diathermy of carcinoma, sarcoma, endothelioma and fibrosarcoma of the upper air passages and attempt to illustrate the benefits of the diathermic treatment in our struggle with these malignant tumors.

We all know that now and then we come across malignant tumors in the upper air passages with a relatively good prognosis, because we, owing to an early diagnosis and energetic operative treatment, are able to cure these patients.

In spite of these good results obtained by surgical and radiotherapeutic treatment, there are many cases of malignant tumors which are not recognized in time by general practitioners and therefore come too late under special treatment. I am especially thinking of malignant tumors in the jaw, the roof of the pharyngeal nasal cavity or in the lower part of the pharynx and in the larynx. These tumors are generally too advanced when they come under special treatment and the chances for cure are very small, even if extensive, dangerous and mutilating operations are performed. It is especially in these cases that we see the benefit of surgical diathermy, and even if a cure cannot be obtained the electrocoagulation is able to alleviate the pain and ease the patients.

A surgeon who for the first time has the opportunity of seeing diathermy used in these distressing cases which up to now have been considered inoperable, will be surprised at the good results that can be obtained, and as Dan McKenzie observes, it most forcibly leads him to regard diathermy as the most merciful agent in the treatment of this distressing form of cancer, with which science has supplied him." (See Acta Otolaryngologica, Vol. VII, 1925, p. 600.)

I will now illustrate the results of the treatment of neoplasms from different regions. As to the instrumental and physical part of the treatment I have to refer you to the modern textbooks and to the latest volumes of Acta Otolaryngologica.

THE DIATHERMIC TREATMENT OF MALIGNANT TUMORS OF THE NOSE AND THE UPPER JAW.

Professor Gunnar Holmgren, of Stockholm, has, during the last years, especially taken up this treatment in cases of malignant growths in these regions and I quite agree with him that the results gained by electrocoagulation of this kind of malignant growth are better than those gained by ordinary surgical treatment. This is especially the case with tumors not extending outside the jaw, as a successive destruction and extirpation of the tumor by means of electrocoagulation eliminates every possibility of a contagious and metastatic spreading of the disease and thereby prevents recidive.

But even in the inoperable cases one can obtain fine results by electrocoagulation, which I am going to illustrate by giving you the history of a 44-year-old man who suffered from a fast-growing and quickly-recidivating *sarcoma of the upper jaw,* which before the diathermic treatment would have been considered hopeless and inoperable.

This patient has for three months suffered from a bleeding tumor, which entirely blocked the right side of his nose. On Aug. 23, 1926, one of my assistants, during my absence, attempted to remove the growth by means of Dencker's operation for empyema of the upper jaw. The sinus maxillaris was filled with bleeding neoplasm which had perforated the anterior wall, the roof and the posterior wall of the cavity, and the ethmoid cells were also involved.

The operation could not be completed and after a series of treatments with strong X-rays, he left the hospital Sept. 6, but had to be readmitted Oct. 27, as the tumor was growing fast and he suffered from pains in the right side of his head and had a fetid secretion from the nose.

Oct. 28 he was perorally intubated under chloroform narcosis and his right upper jaw was exposed by means of an ordinary resection-incision. The anterior wall was destroyed with a diathermic knife, the content of the cavity was coagulated with a ball-shaped electrode, whereupon the coagulated tumor was removed with sharp spoon. The part of the tumor which extended blackwards into the choana posterior was removed in the same way. The facial wound was closed and the cavity of the jaw and nose was packed with idoform gauze.

The patient felt very much relieved after the operation, but a recidivation set in very soon after, with pains and bad-smelling discharge and he was again admitted to the hospital Dec. 29, 1926. There was no tumefaction of the glands.

On Dec. 30 he was again operated in the same manner, peroral intubation and reopening of the facial wound. But this time I removed by means of diathermy the right half of the hard palate, together with processus alveolaris and its teeth, so that the buccal cavity and the cavity of the jaw and the nose formed one single cavity which could easily be kept under observation. The enormous growth could now be thoroughly removed by electrocoagulation and the main part of the growth, which extended backwards into the nasopharyngeal cavity, was now easily destroyed.

The after-treatment passed without reaction, except that he got an erysipelas facies two-and-one-half weeks after the operation.

During the nine months that have elapsed since the operation he has been quite well and has been able to do his work as a railway official. By removing his dental prothese, which covers the great defect in his palate, the whole cavity can be examined; it is covered by smooth and healthy mucous membrane. His speech is quite natural when the prothese is in position, and the cosmetic result is excellent.

Having performed a number of similar serious diathermic operations, one is surprised to observe how little the patients react even after a very lengthy and energetic treatment.

First, it is my opinion that it is best for the patients to keep them in a deep narcosis during the operation in connection with peroral intubation.

When diathermy is used in the upper air passages chloroform must be used, as ether may be dangerous on account of ignition.

To prevent short circuiting by contact of the electrode and the intubation tube the latter has to be covered with an India-rubber tube.

A diathermic treatment of malign growths in the nose and upper jaw takes a long time, from two to three hours. The operator must take his time, not get tired, and proceed very slowly so as to work down deep and remove all the malignant neoplasms. These have to be thoroughly coagulated and then removed with a sharp spoon, and in this manner you work through until you reach the healthy tissue.

The shock of the operation is surprisingly small, and old patients (I have operated patients up to 80 years of age) can very well stand the long diathermic treatments of extensive tumors of the upper jaw; they develop no fever and can get up a few days after the operation.

To get a satisfactory result in malignant growths of the upper jaw it is best always to remove the hard palate with the alveolar process so as to gain a free view of the field of operation. I did not do this

the first time I operated on the patient I have just mentioned, and the result was a quick relapse, which did not occur after the second operation, where I removed the whole roof of the mouth. In case of recurrence the large defect in the roof of the buccal cavity will make it easy to discover it and repeat the treatment.

Cosmetic considerations ought never to have any influence upon the extent of the operation, and if the growth involves the orbita or the skin of the face the whole content of orbita, including the eyeball and part of the face, must be removed.

Defects of the face cause by this operation may later on necessitate plastic operation so as to hide the defects.

There is still another circumstance in the case of the last-mentioned patient which I should like to dwell upon, namely, the attack of facial erysipelas that developed about three weeks after the operation and which might possibly have had some influence on the good result.

You all know, I am sure, that some years ago it was claimed that sarcoma could simply be cured by infecting the patient with erysipelas. I have often tried to infect patients, suffering from malignant tumors, with erysipelas but have always failed. On the other hand, several years ago I happened to treat a young man from Iceland, who suffered from an incurable sarcoma of the upper jaw. Shortly after I had attempted in vain to remove the growth, he got a facial erysipelas, whereupon the sarcoma disappeared of itself, and he has since then, according to reports from his physician, been perfectly well.

I will not dwell much longer on the diathermic treatment of malignant tumors of the nose and jaw, but only remark that I have employed surgical diathermy in the case of a woman, age 71 years, who had an extensive destructive endothelioma in the upper jaw, and in the case of a man, age 63 years, who had a carcinoma in the upper jaw. In both patients the tumor had caused an enormous tumefaction of the buccal cavity.

In both cases one could, one-and-one-half years after the operation, examine the field of operation through the defect of the palate, and verify that there was no relapse.

On patient No. 4, a man age 62 years, who suffered from a solid carcinoma of the right upper jaw, the jaw was removed and ten months later, on account of recurrence, the right eye was removed. About five months after the last operation he was well and the cavity looked healthy.

In Case 5, a man age 38 years, the carcinoma of the upper jaw was so pronounced that the diathermy was of no use.

In Case 6, a man age 67 years, with a solid carcinoma, there was no relapse a month after operation. Have not seen him since.

In Case 7, a woman age 66 years, with a very advanced adenocarcinoma, the operation gave excellent results, but there was a relapse seven months later.

In Case 8, a man age 79 years, with an immense carcinoma of the upper jaw, the electrocoagulation gave a good result; no reaction; he was out of bed a few days after the operation, but had a relapse some months later.

It is of course impossible to give a decided opinion of a treatment only used for a few years and having had the opportunity to observe only eight cases of malignant tumor of the upper jaw.

Considering the unsatisfactory results of general surgical treatment in cases of sarcoma and cancer of the upper jaw, and that amongst my eight cases three were cured by using surgical diathermy, I do not doubt that we in electrocoagulation possess the most active, and at the same time most merciful and least disagreeable method for destroying malignant growth of the upper jaw.

I prefer it to the radium treatment, which is very uncertain in its effect and is often followed by slow necrosis of the upper jaw.

By two patients who suffered from carcinoma of an upper jaw, a man age 54 years, and a woman age 37 years, I obtained a cure without recidive by using up to 80 millicuries, introduced for three days in the cavity of the jaw, but it took about two years before the necrosed jaw was expulsed and the patients suffered greatly during that period.

I mention these two cases because I wish to point out that X-ray treatment, connected with resection, result in cure, but I believe that the chances in this respect are better if surgical diathermy is used from the beginning.

By means of electrocoagulation the lymphoid vessels are closed and there is much less danger of metastatic infection and spreading of the tumor by contagion than if the tumor is removed by a bloody operation.

Personally I have not had the opportunity of using electrocoagulation on *malignant growths in the nasopharyngeal cavity,* but according to cases, referred in literature, the method has given good results.

I have, however, had very fine results with radium treatment in cases of cancer in the nasopharyngeal cavities, and as this treatment

is easy to carry out and the results good if sufficiently big doses are employed, it may be doubtful whether electrocoagulation can or ought to replace radium in these cases.

If, however, electrocoagulation is preferred, I believe that we ought always to procure free access to the nasopharyngeal cavity by diathermic perforation of the soft, eventually also of the hard palate, so as to get a free view of the field of operation.

I have only once had occasion to employ electrocoagulation, in the case of a young man age 17 years, who suffered from a large *fibroma of the nasopharyngeal cavity*, which I had treated in vain for three or four years.

As a rule we attack these fibromes through the nose and the upper jaw in deep narcosis and after peroral intubation.

In this case, however, the tumor adhered so to the walls of the nasopharyngeal cavity and the bleeding was so violent that the attempt to remove it had to be given up.

He was subjected to a series of electrolytic treatments, of up to 200 milliampere current power, without much effect; and it was only after a series of diathermic treatments during the last two years that the tumor has begun to decrease. The diathermic electrodes were partly introduced through a defect in the soft palate, partly from the front through the nose, and partly guided up to the tumor behind the soft palate. He is now much better, has good passage through the nose, and I believe that diathermic treatment is to be recommended in such severe cases of nasopharyngeal fibromes which cannot be removed by an ordinary surgical operation.

Buccal carcinoma has been successfully treated by electrocoagulation and there is already a quite important literature on the subject, according to which surgical diathermy has proved to be a good remedy for cancer of the cheek and buccal cavity.

According to Elis Berven, of Stockholm (Die Behandlung des Bukkal-Carcinomes, Acta Otolaryngologica, Vol. VII, 1925, p. 526), who has treated a great number of buccal tumors partly with radium and Roentgen, 49 cases, and partly with electrocoagulation, 19 cases, he has come to the conclusion that it is doubtful that in these cases a real cure can be obtained by radium and Roentgen treatment, whereas electrocoagulation seems to give the patients a very good chance.

These forms of cancer are apt to give metastasis of the glands, and Berven points out the advantage given by electrocoagulation in this respect, as it seals the blood and lymph channels and thus lessens the likelihood of local recurrence and metastasis.

The result of the operation is best in such cases where the buccal cancer has not caused visible metastasis of the glands. Clark found that a recurrence always occurred in case of glandle metastasis, whereas he, amongst 14 cases of localized buccal cancer, had eight cases which were free from symptoms after two years.

In 19 of Berven's cases, treated with electrocoagulation, nine had no pronounced glandular metastasis; of these, six are free from symptoms, while nine patients who at the time of the operation had evident glandular metastasis, all died, a proportion which, Berven writes, covers the experiences of Clark.

My own experience is only supported by two cases. The first, a woman age 56 years, in whom an enormous cancerous tumor had developed in the course of one month. It extended from the right

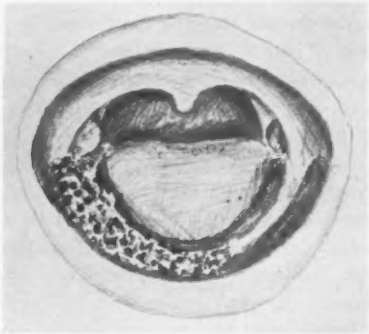

Fig. 1. Woman, aged 69 years. Buccal cancer.

sulcus alveolobuccalis and involved the whole maxillary angle and had extended through the cheek, where there was found an ulcerated tumor of the size of a child's head; internally it had involved the root of the tongue. There were no visible metastases of the glands. The tumor was in deep narcosis, easily removed by electrocoagulation, the lower jaw was to a great extent destroyed by cancer. After the operation was finished, the tongue and pharynx could be seen through the large opening in the cheek. There was no bleeding during the operation. To render the intake of nourishment possible, a sonde a demeure was introduced into the esophagus through the nose. In spite of the very extensive operation, the condition of the patient was excellent. The horrid smell from the wound disappeared at once; the temperature was normal, but on the fourth day after the operation she died suddenly from heart disease.

The other patient was a woman age 69 years, with an ulcerating cancer of the right side of the lower jaw. The posterior end reached the right tonsil and extended forward to the middle line covering both sulcus alveolobuccalis and lingualis. In one place it went over unto the tongue. The tumor had developed in one-and-one-half years and was very tender to the touch. No infiltration of the glands (see drawing). Microscopical examination: Carcinoma solidum. April 8, 1927, deep narcosis, electrocoagulation. A month later every trace of the tumor had disappeared. A sequester corresponding to the upper ridge of the jaw was in process of expulsion. A local recidive had to be electrocoagulated four months later.

I have never had an opportunity to treat malignant neoplasms of the tonsils with electrocoagulations.

On the other hand, I have in the course of years removed both cancers and sarcomas of the tonsils by means of general surgical excision, with very good results. There is no doubt, as also pointed out by Gustav Spiess, from Frankfurth, and other authors, that electrocoagulation can be employed with good results in cases of localized tumors of the tonsils.

In cases of extensive ulcerated malignant growths that extended beyond the boundaries of the tonsil into the soft palate and the root of the tongue, it is safer to refrain from operative surgery which gives bad results, whereas electrocoagulation, after splitting up the cheek to give room, gives good chances for cure. In such cases I have also had satisfactory result by using radium emanation in glass tubes introduced into the growth.

Opinions vary very much as to the treatment of cancer of the larynx with electrocoagulation. Several authors have expressed as their opinion, that one ought to be very careful in employing electrocoagulation in the larynx, as it may easily cause serious reaction, followed by stenosis, and may also give rise to necrosis of the cartilage of long duration.

I wish here to remark that I have repeatedly used electrodiathermy in cases of malignant tumors of the larynx without yet ever seeing any of the above-mentioned complications, and the reaction after electrocoagulation has been very slight and of short duration.

I first call attention to the difficulties the surgeon meets every time he has to decide in cases of larynx cancer if the case can be treated at all, and if so, how to treat it. It is quite natural that a general surgical treatment ought to be used in cases of cancer which are recognized at an early stage of their development and are so situated that they are easily accessible.

For instance, I doubt that any modern laryngologist in a case of localized cancer of the vocal cord could recommend a better treatment than a thyrotomy with excision of the diseased vocal cord.

Should, however, a cancroid of the vocal cord be so extensive that one is not sure of being able to remove it thoroughly by a simple thyrecotomy and excision of the diseased vocal cord, the question will be, should we make a total resection of the larynx, or should we try to destroy the endolaryngeal cancer by means of electrocoagulation, or possibly try the doubtful destructive effect of radium in connection with Roentgen rays, or should we leave it alone, only performing a preventive tracheotomy.

Early diagnosis of the cancer is of the uttermost importance in deciding which treatment one ought to employ.

There are periods in the development of a neoplasm, where one is doubtful as to whether one should operate in the general surgical manner or not, and here the technical skill of the operator, combined with his lesser or greater experience, is of the greatest importance in choosing the right procedure. Mistakes are often made in this respect.

I remember I operated on a woman, age 40 years, about ten years ago, who suffered from an extensive cancer in the region of the right arytenoid cartilage and sinus pyriformis. The tumor was so large that I was convinced that it could only be removed by means of a total resection of the larynx. The patient would not permit this operation. I therefore restricted the operation to a preliminary tracheotomy, followed by a pharyngotomia subhyoidea and lateralis, through which I removed the tumor as thoroughly as possible. It is now more than ten years since the operation, and the patient is perfectly well.

I believe that for certain forms of cancer surgical diathermy is an excellent treatment, which not only eases the pain and lengthens the life of the patient, but it can also cure patients suffering from a cancer which is so placed in the larynx that ordinary operative treatment would necessitate serious, dangerous and very painful operations.

I am thinking of such cases in which there is a cancer on the epiglottis or aryepiglottic fold, reaching down into the sinus pryriformis. Even if in such cases it is made possible, by means of subhyoide pharyngotomy, to get at and extirpate the tumor, there is a great risk for the patient in the seriousness of the operation itself, the danger of inoculation is very great, because parts of the tumor may touch and infect the wound, and finally the probability of metas-

tatic infection through the blood channels makes the result very problematic.

Cancer of the upper aditus laryngis and sinus pyriformis form one of the saddest chapters in the history of laryngological operations, and the footprints warn us. Fortunately these cancers belong to those diseases of the upper air passages which can be treated with comparative ease by means of electrocoagulation, and the experiences I have had on this subject confirm my belief that we, in electrocoagulation, have found a method which under certain circumstances can be of great benefit to these patients.

Electrocoagulation in cases of cancer in the upper part of the larynx, epiglottis and sinus pyriformis, can be used in indirect laryngoscopy by means of adequately bent electrode, preceded by local anesthetics. Personally, I prefer deep chloroform narcosis and suspension laryngoscopy. This method is also recommended by Dr. Frank I. Novak, of Chicago, who has written about electrocoagula-

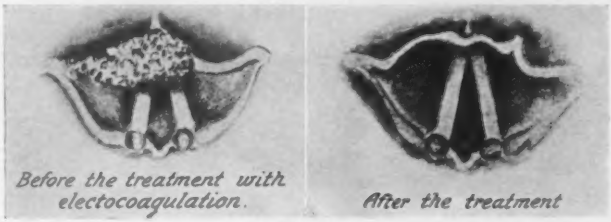

Fig. 2.

tion of malignant laryngeal tumors under suspension laryngoscopy in Acta Laryngologica, Vol. VII, 1925, p. 626.

In cases of lower situated cancers of the larynx it is better first to do a thyrectomy and through this free entrance to the inner part of the larynx perform the electrothermic operation.

I will now give you some personal observations of cancer of the epiglottis, which I have destroyed by surgical diathermy.

My first case[1] was a man, age 57 years, suffering from an extensive cancer of the epiglottis. The disease had lasted for three-fourths of a year and the symptoms were increasing difficulty in swallowing, pains radiating to the right ear. The right side of the epiglottis was occupied by a white spongy tumor. No enlargement of glands. The patient suffered from chronic bronchitis, a weak heart and had on account of drink not much vital resistance.

Sept. 24, 1925, I did a preliminary tracheotomy as I feared that the electrocoagulation might cause a reactionary swelling and stenosis

of the larynx. Nothing of this sort happened. Oct. 15, under chloroform narcosis the patient was placed in suspension laryngoscopy, and the tumor destroyed by a ball-shaped electrode. The after-treatment gave no difficulties. The pains disappeared in the course of two weeks, and by laryngoscopical examination three weeks after operation the tumor was found to be expelled and there was a large cicatricial defect of the epiglottis, as demonstrated by the figures before and after the treatment.

During the following week no trace of tumor could be found, but after the patient had been out of his bed for six weeks his heart failure increased and he died suddenly from heart-collapse Nov. 29, 1925.

This observation showed the advantage of surgical electrocoagulation of a large cancer of the epiglottis which was radically removed by a single electrodiathermic operation.

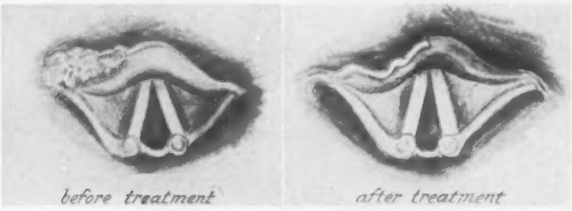

Fig. 3. Man, aged 54 years.

The second case of cancer epiglottis came under my treatment May 14, 1926, at the university clinic.[2]

The 54-year-old man had, as shown by figure, an uneven nodular not distinctly ulcerated neoplasm, which occupied nearly the whole right half of the epiglottis, stretching into the right pharyngoepiglottic fold and continuing out into the lateral wall of the pharynx. Microscopical examination showed: Carcinoma. No swelling of the glands. May 15, 1926, electrocoagulation and chloroform narcosis in suspension laryngoscopy. The strength of current was ½ milliampere. The patient was perfectly well after the operation, no local reactions, no difficulties in breathing.

A month later the patient was perfectly well, no difficulties in swallowing. By laryngoscopy was found a nearly total defect of one side of the epiglottis.

The lateral part of the defect showed signs of a local recidive. I therefore put him under chloroform again and repeated the electrocoagulation of this suspicious lateral part of the defect.[3] The

patient has since been well and comes now and then to be examined; seen last a year after the operation.

These two cases are good examples of the advantage of the electrodiathermic treatment of these forms of cancer laryngis. In the first case a single operation was sufficient to remove the tumor, whilst in the second case it was necessary to repeat the operation a month later with good result.

If the seat of the cancer is extralaryngeal, *i.e.*, on the posterior wall of the larynx or in sinus pyriformis, it will nearly always be necessary to destroy the cancer in several seances, because the tumors as a rule are more extensive than they seem to be when seen as in a birdseye view by means of indirect laryngoscopy.

These tumors are always diagnosed too late and are therefore far advanced in development when they come under treatment.

I have lately treated an old lady of over 80 years with a cancer of sinus pyriformis. In spite of her age and of a pronounced heart disease this old lady stood the electrodiathermic treatment very well

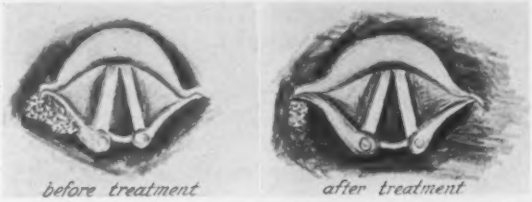

Fig. 4. Woman, aged 80 years. Cancer of disinus pyriformis.

indeed. The disease had given her symptoms for three-fourths of a year, pains irradiating to the right ear. Swallowing was free. On the right side of the neck slightly swollen glands could be felt. Laryngoscopic examination showed, see figure, at the back of the right aryepiglottic fold a large grayish ulcerating tumor extending down into the sinus pyriformis and out to the lateral wall of the pharynx. The mobility of the right side of the larynx was diminished.

What ought one to do in a case like this, an 80-year-old woman with a weak heart? To remove the tumor by means of an extensive external surgical operation was out of the question. Radiotherapy gives unsatisfactory results in cancers of these regions.

One could either do nothing and let the patient succumb slowly or try by means of the electrocoagulation to eradicate the tumor, eventually relieve the patient for a time. She chose the latter and on June 2, 1927, she was chloroformed and in suspension laryngo-

scopy the tumor was easily removed by electrocoagulation. The whole operation, including the narcosis, took only 15 minutes. With the exception of slight pains in swallowing during the first week, she felt perfectly well, and there was no reaction from the larynx. Three weeks later the electrocoagulation-crust had disappeared. She now felt quite well and was up all day. Six weeks later slight remains of the tumor was seen on the lateral wall of sinus pyriformis. I therefore repeated the same operation on July 11, 1927. Two days later she was out of bed and returned to her home in the country three days later.

I show you an epiglottis-cancer in a 40-year-old man, which is still under treatment, and where the whole epiglottis was destroyed by means of electrocoagulation.

Of course the time of obseravtion has been too short to judge of the final result, but this case proves that one can electrocoagulize extensive cancerous affections of sinus pyriformis, even on patients

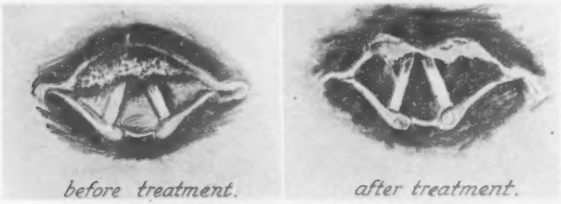

Fig. 5. Cancer epiglottidis.

80 years old, and give them a chance to be cured of a disease which must otherwise be considered fatal.

I have had the opportunity of treating in the same manner even larger cancerous neoplasms in sinus pyriformis, which extended to the root of the tongue and tonsils. We had here to do with tumors which were inaccessible to surgical treatment, and these were by means of a series of electrocoagulation considerably reduced and the patient was relieved from pains and capable of working for a time, even if incurable.

Finally, considering endolaryngeal cancers, my opinion is, as already remarked, that in cases of localized cancroids of the vocal cords, one ought always to excise the affected vocal cords through a thyrectomy, as I do not believe that any other method can give better results. Should, however, the tumor have extended outside the vocal cord a surgical excision would effect a cure. The question is, therefore, should one in these cases make a total resection of the larynx or depend upon electrocoagulation after thyrectomy?

It is impossible to give decided rules as to how these forms of endolaryngeal cancer should be treated, but I believe, that if the patient is a strong, healthy individual, his best chance is a total resection. The results I have obtained by this method are so good, presuming of course that there are no recidives or metastasis, that in suitable cases I would always try a total resection, the immediate mortality of which is greatly reduced by our modern methods of operation.

In cases of very extensive cancer of the larynx in individuals who are unsuitable for operation on account of corpulency, short neck, weak heart or other weaknesses, I believe that the electrocoagulation gives the best results. It ought also to be employed in such cases where the patient objects to the operation on account of the immediate risk.

Last year I had opportunity to employ the method on a man, age 58 years. He had for about two years suffered from increasing

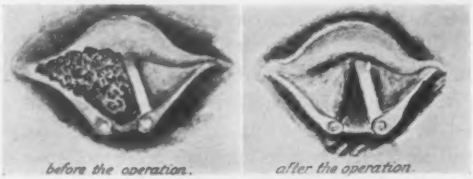

Fig. 6. Epithelioma laryngis.

hoarseness, but had only lately consulted a specialist, who sent him to me. He had no difficulties of respiration, was quite aphonic, no difficulties in swallowing, and felt otherwise quite well. The patient was a very large and powerful man, very broad and fat. Laryngoscopy shows, see figure, an uneven, knotty, partly ulcerated tumor, filling the whole right side of the larynx, which was fixed. The principal part of the spongy tumor included the right vocal cord and ventricular band and extended up to the epiglottis and out to the right aryepiglottic fold. The left side of the larynx was normal. Nothing abnormal could be felt on the outside of the neck and there was no metastasis of the glands.

The question was now, should one propose a total extirpation of the larynx, or be contented with electrodiathermy and a palliative tracheotomy?

The patient was very fat and short-necked, so that the prognosis of a total extirpation was not good; on the other hand, the neoplasm

was so extensive that there was no possibility of removing it radically through a laryngofissure.

I decided to try electrocoagulation, which was carried out in two seances after previous tracheotomy in local infiltration anesthesia. A fortnight later, March 5, under chloroform and in suspension laryngoscopy the tumor in the upper aditus of the larynx was removed by means of energetic electrocoagulation. Three weeks afterwards, March 26, laryngoscopic examination showed: the crusts had disappeared, deep down on the right real and false vocal cords one could observe remains of the tumor; these were removed by electrocoagulation after laryngofissure under local infiltration anesthesia.

The patient left for home five weeks later, May 6, 1926, and examination showed that the tumor had nearly disappeared and the cavity of the larynx was wide and free, see figure.

I have not had the opportunity of examining him again, but his physician wrote me a year later, May 17, 1927: "His general health is good, there is good passage through the larynx; for safety's sake, he, however, carries a tube. He is very grateful for your excellent treatment, which has given such an excellent result."

All is well so far; but for private reasons the patient could not be persuaded to come over and be re-examined so as to make sure if there were still remains of the tumor or not. Should this be the case it would be easy to treat him again with electrocoagulation.

I am afraid that what I have told you is neither new nor of special interest to my American colleagues.

Many continued observations must be made before we can come to a decided opinion as to the importance of electrocoagulation as a weapon in our struggle with the tumors of the upper air passages.

The more our technic is developed, the earlier these malign neoplasms come under treatment, and the more experience we gain, the better will be our results.

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2. Published in Acta Otolaryngologica, Vol. X, 1927.
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THE HOARSENESS OF INFANTS AND AN EXAMINATION INTO ITS CAUSES.

Dr. Taro Matsui, Mukden, China.

There are many cases of infantile beriberi which attacks infants who are nourished with the milk of a mother who is suffering from beriberi, and hoarseness is one of the important early symptoms of the disease.

The statistics of Dr. Miyake are as follows: *1.* Number of patients, 262; with hoarseness, 135 (51.5 per cent); without hoarseness, 127 (49.5 per cent). Aphonia existed in 1.9 per cent of all cases. *2.* Appearance of hoarseness as the first symptom, 66.7 per cent; in the course of the disease, 33.3 per cent.

Prof. Hirota found hoarseness in all of his 52 cases where the mother or nurse suffered from beriberi; therefore, in Japan it is an important matter to investigate hoarseness in a small child. Prof. Ino Kubo was the first man who examined the larynx of infants suffering from beriberi. He examined his cases with Killian's tube in the direct method and ascertained that the hoarseness was caused by paralysis of the recurrent nerve, as in the case of an adult, and by edema in the larynx. This paralysis appeared mostly on the left side and disappeared with the cure of the beriberi.

It was already discovered by Professors Baelz, Aoyama, Miura and Okada that paralysis of the recurrent nerve comes in beriberi but their cases were adults, and hoarseness is not an important symptom of beriberi in adults, appearing more seldom and mostly during the course of the disease, along with other usual symptoms.

On the contrary in an infant it comes very frequently and often early as the only symptom of beriberi. In my article, "Direct Laryngoscopy of Small Children and Babies", there are noted six cases of infants with hoarseness, and in five cases the hoarseness was caused by paralysis of the recurrent nerve, due to beriberi, and only one was from laryngitis.

I have seen many cases which had only hoarseness, or aphonia with or without cough, and upon ascertaining that the recurrent nerve was paralyzed, I ordered as the first and most important thing the giving up of the mother's or nurse's milk. This was done and the disease cured. I have had 16 cases of hoarseness in infants during the last

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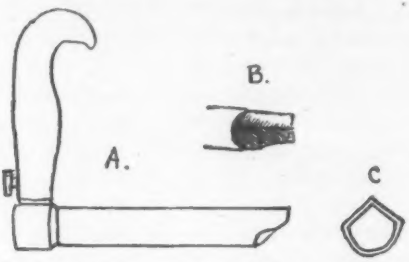
two years. All of them were laryngoscoped, with the following results:

In 16 cases the cause of hoarseness was paralysis of recurrent nerve, 12; acute laryngitis, 3; acute subglottitis, 1. In 12 cases of paralysis of recurrent nerve: left side, 11; right side, 1. Hoarseness appearing as the only symptom, 6; with other symptoms of beriberi, 6. Food: *a.* mother's milk, 11; *b.* artificial food, 1. Beriberi in mother: early or present, 4; none, 7.

As the table shows, most cases of hoarseness of infants in Japan are caused by paralysis of the recurrent nerve, due to beriberi, and a small number from other diseases of the larynx. Paralysis generally appears in the left recurrent nerve, the right side seldom being at-



(A) Tube; length, 11.5 c.m.; diameter (inside), 1.5 c.m. (B) Top from inside. (C) Cutting surface.

tacked; this is due perhaps to the difference in length of the two nerves. In adults beriberi commonly attacks first the peripheral nerve of the sole of the foot, which is the longest nerve in the body.

It can be said in my cases that beriberi has some relation to the season. There were one in April and May, five in June, two in July, one in September, two in October, two in November, and one in December; in January, February and March there were none.

The examination of the larynx of an infant is attainable only through the direct method. Prof. Kubo used Killian's tube in his cases, and Dr. Jackson designed his tube for children, but both must be used under electric light. I use a tube of my own design (see figure), which can be used even in daylight, with a reflector, and it does not slip when pressed on the middle of the tongue, thus a better view can be obtained.

I always examine without an anesthetic; the best position of patient for examination is lying on the back with the head projecting beyond the end of the examination table.

ANGIOSARCOMA OF TONGUE; CASE REPORT.*

DR. JOHN H. HARTER, Seattle, Wash.

Mr. A. S., age 53 years, white, German descent, single; came to office April 18, 1927.

History: Patient on his way to Seattle two days ago visited Dr. Thomas W. Kelsey in Spokane, who referred him to me for diagnosis and treatment.

Has had two papules on tongue for about 30 days; one of these appeared about four hours later than the other. Have increased in size. No soreness at any time. General health excellent, except has lost 10 pounds, with some indigestion, during past four months. Has smoked seven cigars a day for the last 30 years.

Examination: On dorsum of tongue at junction of anterior and middle thirds there are two papules about 7 m.m. in diameter. These are slightly raised, blood-red in color, and there is between the papules and tongue a definite encapsulating membrane. By palpation, papules are felt to extend about 5 m.m. down into the tongue. Throat and mouth normal, no increase in salivation. Cervical lymph-nodes not palpable. Temperature and pulse normal.

Blood Wasserman negative. Urine negative. R. B. C., 4,470,000. W. B. C., 10,200. Lymphocytes, 23 per cent. Mononuclears, 4 per cent. Neutrophiles, 72 per cent. Eosinophile, 1 per cent. Hemoglobin, 80 per cent. Reports ruled out syphilis or leukemia.

A small portion of one of the tumors was removed and sent to laboratory, which reported that diagnosis was impossible from so small a specimen, but that the growth appeared definitely neoplastic. Patient seen in consultation by Dr. Stephen Parker and Dr. L. L. Stevens.

Thinking that waiting a few days would do no harm and that these lesions might be a peculiar manifestation of Vincent's angina, I gave him three subcutaneous doses of sulpharsphenamine, 0.4 gm., and three intravenous doses of mercurosal, 0.1 gm.; also gave the patient a salvarsan gargle to alternate with sodium perborate gargle.

After 12 days the only change in the tumors was that each was growing up definitely out of the tongue, forming an encapsulated mushroom-like tumor.

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*Read before the Puget Sound Academy of Ophthalmology and Otolaryngology, May 17, 1927.

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Believing that any further delay was contraindicated, May 7, under local anesthestic, Dr. L. L. Stevens and I excised these growths with a cautery knife, taking a wide margin of tissue at side and beneath the growths. These elliptical areas were closed with interrupted sutures of braided silk. The tumors were sent to the Physicians Clinical Laboratory and the following report was given from there:

Fig. 1. Photograph of tumor

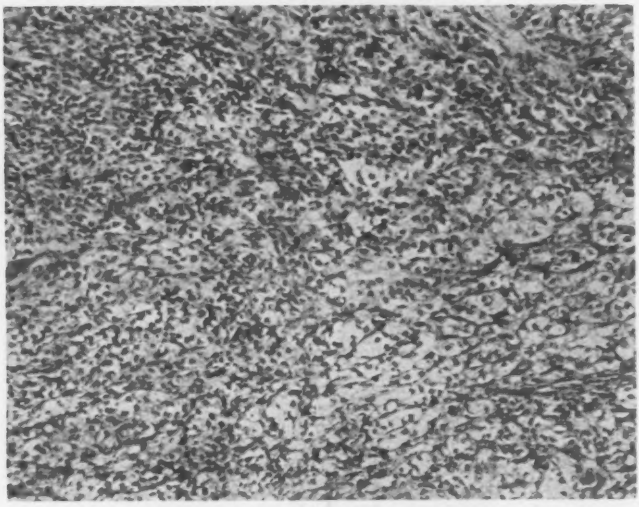

Fig. 2. Microphotograph of tumor

"This is a definite neoplastic process, which arose subepithelially and which has pushed through the epithelial layers. In structure, it is composed of rather large, pale cells, epitheloid in type, with abundant intercellular substance, arranged chiefly in lobulated masses, but in areas showing numerous lumina, some of which contain red blood cells. While the tumor is unusual in structure, the cellular charac-

teristics and arrangement justify a diagnosis of hemangioendothelioma. The growth is invasive and locally malignant. Early metastasis is very improbable.—(Signed) Dr. V. Cefalu."

One of the tumors was sent to the Army Medical Museum, Washington, D. C., from which the microphotographs and the following pathological report were returned:

"Section shows a granulating surface infiltrated with leukocytes and fibrin with a pseudomembranous, but a rather thin layer of exudate on the surface. The upper portions appear to be granulation tissue, densely infiltrated with inflammatory cells. Deeper in, the tissue is made up of a network of cells forming embryonic type of vessels, without any apparent connection with definite lumina. Many of these cells show pale areas or vacuole-like areas, as though hydropic. Fibroblastic structures are numerous, but the cells appear more of the adult type. There are few mitotic figures seen.

"*Diagnosis:* Angioma, or angiosarcoma. It is impossible to determine the exact degree of malignancy, owing to the secondary inflammation.—(Signed) Maj. Geo. R. Callender, Med. Corps, U.S.A."

Convalescence was uneventful. Patient up and about in four days. Has been given X-ray therapy of neck as prophylactic against metastasis. He now, three months after operation, appears well.

These lesions presented difficulties in diagnosis. No similar case report or photograph was found in a search through the literature. The consulting physicians and pathologists had seen no similar lesion. The growths being multiple, malignancy was not at first strongly suspected.

This case is reported in full in the hope that it may be of some aid in the diagnosis of this rare condition.

709 Cobb Building.

POSTOPERATIVE TONSILLAR HEMORRHAGE DUE TO THE ORGANISMS OF VINCENT. REPORT OF FIVE CASES.

Dr. Albert P. Tibbets, Washington.

Postoperative tonsillar hemorrhage from infection of the fossae by the organisms of Vincent is of comparatively rare occurrence. In an active clinical experience over a period of 15 years, we have recognized this condition but five times. Strangely enough, all five cases have been observed within the last two years. While it is quite possible that some few cases of persistent postoperative bleeding in the past have been due to this type of infection, we do not recall any previous case that presented the characteristic symptoms which we have learned to associate with Vincent's infection.

The early recognition of this type of infection is extremely important, for the reason that the treatment is essentially different from that usually employed for postoperative hemorrhages. The patient may be saved much discomfort, and the surgeon considerable anxiety, by the early application of specific remedies.

Etiology: Of the five cases to be reported, three were males and two females. All were young adults. Only one had had military service, having served in the navy. One girl's brother-in-law, who lived with the family, had had two attacks of trench mouth, for which he had been treated by his dentist. In the other four cases there was no definite history of exposure.

In all five cases both the spirilla and fusiform bacilli were recovered in sufficient numbers on direct smear to leave no doubt that they were the specific cause of the necrosis and ulceration which led to the bleeding.

Symptoms: There has been a striking similarity in the symptoms of these patients. All complained of repeated attacks of tonsillitis, and of soreness and discomfort in the throat upon the least exposure to cold and dampness. They were all slightly under weight, anemic, easily tired, suffered from muscular pains, which at times were quite severe, and complained of an excessive amount of thick, stringy mucus from the membranes of the upper respiratory tract. Those seen following an attack of tonsillitis appeared prostrated out of all proportion to the severity of the attack, so that operation had to be postponed from two to eight weeks.

The postoperative symptoms were much the same in each case. In one patient the bleeding began five hours after operation, and in the others from two to seven days. Those patients in whom the bleeding started after 48 hours did not appear to be doing well. They complained of unusually sore throats on the day before the bleeding started, could not take nourishment and seemed prostrated and mentally depressed.

The hemorrhages came on suddenly, without apparent cause. In two cases from 250 to 500 c.c. of blood were lost within a short time. The blood clotted readily, filling the fossae, but there was a steady trickle from beneath the clot. In all cases there was a tendency for the spontaneous arrest of the bleeding at one point, only to have it break out at another, and then recur at the original site.

Pathology: One patient had been discharged as cured of Vincent's angina two months before. Examination a few days before operation showed the membrane paler than normal and the tonsillar crypts filled with whitish masses, together with a thin, grayish pus. In the other cases thin pus could be expressed from the crypts, and the membranes were paler than normal, though at the time no special significance was attached to the appearance of the throats.

Following the hemorrhage and after the fossa had been cleared of clot, numerous brownish elevated spots about 3 m.m. in diameter could be seen standing out in marked contrast to the pale floor of the fossa. Upon manipulation these spots would break down and bleed sharply for a few moments. The bleeding would stop spontaneously at one point, only to break out at another. Upon attempting to seize these spots with hemostats it was seen that the tissues were necrotic and manipulation only caused more profuse bleeding. Later some of these spots coalesced and a grayish membrane formed. As the disease progressed, the edges of the pillars and soft palate became involved in a shallow ulceration with irregular borders, which bled freely upon being touched.

As the fossa healed, the membrane became less dense and fissured, although it maintained a grayish, bloodless appearance for from 10 to 14 days after treatment was begun.

Prognosis: While there were no fatalities in this series, one of the patients was rapidly passing into an alarming state of exhaustion before the cause was recognized. This was due to the repeated hemorrhages, loss of rest, inability to take nourishment, and the prostration which accompanies the disease. Once appropriate treatment had been started, improvement was rapid and the throat usually completely healed at the end of two weeks.

Treatment: The most effective treatment in our hands has been to rapidly cleanse the fossa of clot, and make firm pressure with a gauze sponge wrung out in pure hydrogen peroxide. This usually checked the bleeding promptly. A string sponge moistened with hydrogen peroxide was then placed in the fossa and left for several hours. It was then removed and the fossa painted with a 10 per cent solution of neoarsphenamine in glycerin every four hours.

Every two hours the throat was gargled with a 50 per cent solution of hydrogen peroxide, followed by a half-saturated solution of sodium perborate.

Under this treatment the pain and bleeding rapidly subsided and the patient's mental and physical condition improved in a remarkable manner.

Case 1: Miss M. O'D., white, age 22 years, was referred for tonsillectomy Feb. 14, 1926. She complained of frequent sore throats and a feeling of lassitude and aching in muscles and joints.

Examination showed the tonsils considerably hypertrophied, and a thin, grayish pus could be expressed from the crypts.

As her coagulation time was five minutes and she seemed in fairly good condition, tonsillectomy was performed the following day under gas-ether anesthesia. She left the hospital at the end of 48 hours, and made daily visits to the office for local treatment.

On the fifth day she complained that the right side of her throat hurt so that she could not eat nor sleep, and she seemed quite depressed and despondent.

On the seventh day she phoned that she was having quite severe bleeding. She was brought immediately to the office, where it was found that the right fossa was filled with a large clot. This was cleaned out and the bleeding stopped by the application of hydrogen peroxide. It was then observed that there was a shallow, irregular ulceration extending out over the pillars. A smear showed the presence of Vincent's organisms in large numbers. Applications of 10 per cent arsphenamine in glycerin were made every three hours. There was no further bleeding and she was discharged as cured one week later.

Case 2: Mr. W., white, age 25 years, consulted me on Nov. 5, 1926, complaining of repeated sore throats, vague muscular pains and an excessive secretion of mucus from the nose, throat and bronchial tubes. He had been under treatment by his family physician, who had referred him for tonsillectomy.

He gave the history of having been operated on for chronic appendicitis at the Naval Hospital in Philadelphia three years before. Two

days after the operation, the wound became very tense, and the doctor removed the stitches and evacuated a large clot. The wound was packed but continued to ooze for several days. In March of this year he had a tooth extracted and the socket bled off and on for a week.

Although his coagulation time was only five minutes, he was given calcium lactate for three days, and 2 c.c. of coagulin on the day of operation. The coagulation time was then four minutes.

A local tonsillectomy was performed on Nov. 9. Throughout the operation there was a gurgling of mucus in the trachea and the patient coughed up a large amount of thick, stringy mucus.

Following the operation it was remarked that the fossae were dry, smooth and glistening, and that no muscle fibers had been uncovered.

At 6 p. m. the nurse reported that the patient had been coughing up some bright red blood throughout the afternoon. Inspection showed a large, thick clot in the right fossa, with blood trickling from beneath it. The clot was removed and an attempt made to check the bleeding by pressure. This was unsuccessful because of the incessant coughing from the excessive mucus in the bronchii. He was taken to the operating room and given a general anesthetic. The fossa showed several necrotic areas, whereas a few hours before it had been perfectly smooth. These areas tore out when grasped with hemostats, making it difficult to take stitches. Seven sutures were required to stop the bleeding.

The patient spent the following day in the hospital, and as the throat was perfectly dry, was allowed to go home on Nov. 11. He began to bleed again the next morning at about 2 o'clock, and was readmitted to the hospital and again anesthetized. While the anesthetic was being given, the hemorrhage ceased spontaneously. Gentle exploration, however, resulted in profuse bleeding from the upper part of the fossa. There did not seem to be any well defined bleeding vessels and the tissues were so necrotic that it was useless to attempt sutures. A tightly rolled gauze sponge was placed in the fossa and the pillars sutured over it. This controlled the bleeding for about 36 hours, when in spite of the sponge, blood began to appear freely at the upper and lower parts of the fossa. At this time it was noted that there was a rat-eaten appearance to the anterior pillar, with a superficial ulceration. A smear was taken immediately and both the spirilla and fusiform bacilli found. The sponge was removed and 50 per cent hydrogen peroxide used to check the bleeding, followed by painting with arsphenamine in glycerin. Immediate improvement was noted. There was no further bleeding and only

occasional spitting of blood following a coughing spell or the swallowing of food. The painting with arsphenamine in glycerin was continued every three hours, and he was sufficiently improved to return home on the eighth day following the operation.

Case 3: Mr. C. E. B., white, age 25 years, was referred by his family physician for tonsillectomy. He had recently recovered from an attack of acute follicular tonsillitis, which had confined him to bed for a week.

He gave the history of suffering from repeated sore throats, aching in the muscles and joints, and an excessive secretion of mucus from the upper respiratory tract.

This patient appeared so anemic and prostrated that he was referred to his physician for a course of treatment to improve his general condition.

On June 17, 1927, his tonsils and some adenoid tissue were removed under gas-ether anesthesia. He left the hospital two days later, although there was an occasional clot of blood in the mucus from the nasopharynx.

Two days later he had a sudden hemorrhage from the left fossa, amounting to nearly 500 c.c. The fossa was immediately cleaned and packed and the patient removed to the hospital. Upon removal of the sponge, the typical appearance of ulceromembranous angina was evident and this was confirmed by finding numerous organisms of Vincent. Some oozing from both the fossa and nasopharynx persisted for several days, but under applications of neoarsphenamine in glycerin and a peroxide and sodium perborate gargle, he was able to return home on the seventh day after the hemorrhage, and was discharged as cured a week later.

Case 4: Mr. S. McG., white, age 21 years, consulted me in June, 1927, for a Vincent's infection of the right tonsil. It responded rapidly to treatment and he was discharged as cured at the end of a week.

On Aug. 27 a tonsillectomy was performed under gas-ether anesthesia. Two days later bleeding started in the right fossa, and examination showed a grayish fissured membrane with several reddish elevations which bled profusely upon being disturbed. A smear showed the organisms of Vincent present in considerable numbers.

Upon the application of neoarsphenamine in glycerin three times a day, and a peroxide and sodium perborate gargle, the bleeding promptly ceased and healing took place in the usual manner.

Case 5: Miss R. L. W., white, age 19 years, was referred for tonsillectomy, Sept. 5, 1927. She had been operated on for the removal

of tonsils and adenoids at the age 11 years. Since then she had been troubled with nasal obstruction and a postnasal discharge. During the past two years she suffered from aching pains in muscles and joints, and exhaustion which was pronounced that she would have to go to bed every day upon her return from school. She was being treated for anemia by her family physician and had gained in strength.

Examination showed that the upper poles of both tonsils were still present, and contained several crypts from which pus could be expressed. There was a turgescence of the nasal mucosa, but no deflection of the septum nor other abnormality.

Tonsillectomy was performed the following day under gas-ether anesthesia. She left the hospital at the end of 48 hours, but made a rather slow recovery. She felt so weak that she preferred to remain in bed, and complained that her throat hurt so bad that she was unable to swallow or to rest well at night. She was visited daily at her home and her throat painted with iodine and glycerin. At that time there was nothing unusual about the appearance of the throat to account for the soreness.

On the sixth day after the operation she had a rather profuse hemorrhage from the right fossa. She was brought to the hospital, where the clot was removed and the bleeding checked temporarily with hydrogen peroxid compresses. A string sponge was then inserted and left in place. It was found necessary to replace the sponge twice during the night as there was considerable oozing. The following morning a smear showed the presence of the organisms of Vincent in considerable numbers. The throat was painted every four hours with neoarsphenamine in glycerin, and a peroxide and sodium perborate gargle used every two hours. This led to rapid improvement in both her local and general condition. She was able to leave the hospital at the end of four days and her throat was completely healed two weeks later.

1801 I Street, N. W.

THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The annual meeting of the American Academy of Ophthalmology and Otolaryngology will be held in St. Louis, Mo., October 15-19, inclusive. Hotel Statler has been selected as headquarters.

Dr. W. P. Wherry, Executive Secretary.

A CASE OF LATERAL CERVICAL FISTULA.

DR. EELCO HUIZINGA, Holland.

In THE LARYNGOSCOPE of July, 1926, Dr. Rudolph Kramer described a very interesting case of lateral cervical fistula, which from a clinical point of view presented some unusual details. He was able to show the course of the fistula clearly by injecting it with a mixture of bismuth and oil, which was clearly visible in the X-ray photo taken of it. Probably Kramer was the first to describe this kind of injection, which is of so great importance to the diagnosis.

When Kramer's article was not yet published, a similar case came to our clinic, which was examined in an analogous way, though we in our case made use of a French preparation, lipiodol, which has recently gained a great reputation. It seems to us that this lipiodol offers many advantages in cases like this.

Fig. 1.

In the first place its composition lends itself admirably to injection into fistulae. Secondly, it mixes easily with the contents of fistulae, which in our case was of great importance. The fistula was not easy to inject and our first photo shows that only a little lipiodol had accumulated immediately under the skin, but in the photos taken the next day the course of the fistula could be distinctly traced.

The short historia morbi of our youthful patient is the following: V. 3 months old was sent to our clinic in May, 1926, because ever since its birth it had had an aperture in the neck which discharged purulent matter at intervals. It also made a grunting noise while breathing. It was the second child of healthy parents; the first child was perfectly normal. In every other respect this child could be said to be normal too.

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On examination the infant appeared to be in a very good state of nourishment; it had a slight stridor and showed slight retractions of the ribs. Its voice was clear and drinking was easy. On the right side of the neck, a little above the sternoclavicular joint, an aperture was to be seen. This aperture could be traced a good way upwards. By palpation a cord of tissue could be felt along the front part of the sternocleido, which evidently disappeared higher up in the neck.

By pressure on the neck to the right side a small amount of pus was discharged. The fistula could not be injected in the direction of the throat. Neither in the mouth nor in the pharynx anything abnormal could be found and even by direct laryngoscopy no inner fistula-aperture was visible.

Attempts were made to inject the fistula with lipiodol, which failed in the beginning. The lipiodol remained closely under the surface

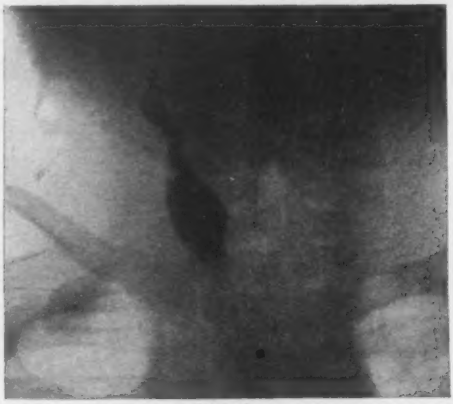

Fig. 2.

of the skin, but the next day the course of the fistula had become clearly visible (Figs. 1 and 2).

The fistula ran parallel with the front rim of the sternocleido with the exception of a few bulgings, went deeper and deeper and ended in the corner of the right jaw.

There was probably an aperture in this typical place under the tonsil, which, however, has not been proved.

The stridor in our case could not be explained with certainty; in the photos it was not clear that a pressure was exercised on the trachea. The movements of the vocal cords were normal. Probably it was a case of so-called innate stridor. Further therapy was given up; the youthful age of the patient made any greater intervention impossible.

Academic Hospital.

SPHENOID SINUSITIS WITH PECULIAR AND UNIQUE SYMPTOMS.*

DR. ROBERT F. RIDPATH, Philadelphia.

Realizing the importance and being cognizant of the difficulties encountered on many occasions in the diagnosing of sphenoidal infection, the subject of this paper suggested itself.

Situated within the body of the sphenoid bone, unequally divided by a bony septum, the sphenoid sinuses occupy a position which, when diseased, may and does give rise to a complexity of symptoms.

Sphenoid sinusitis as outlined by various authors is divided into two classes—that of the acute and chronic varieties. The chronic infection is subdivided again into: *a*. Those with free drainage; *b*. those with deficient drainage.

The various symptoms usually associated with the acute infection, according to Skillern, are subjective, objective and ocular manifestations. These include headache, pain over the mastoid region, inability to concentrate, aversion to mental work, discharge, cacosmia and enlargement of the blind spot, fever, etc.

While the majority of patients suffering from this disease have one or more of these symptoms, others—and we are truly thankful they form the minority—suffer from peculiar symptoms, seemingly unattached to the sphenoid. Some patients make no complaint of headache, while others seek relief only because of pain.

The etiological factors or the history of an infection as given by the patient may be of considerable value in aiding one in the diagnosis of an acute infection, although of very little value or help in a chronic condition.

We must remember that in all acute infectious diseases, the mucous membrane of the upper respiratory tract is involved. That scarlet fever, scarletina, measles, pneumonia, pertusis, influenza and repeated attacks of coryza frequently leave an aftermath in the mucous membrane or tissues adjacent to it. Repeated cultures taken from the nasopharynx in the past year have proven to me beyond a doubt that many of these germs reside in the mucous membrane months after the disease has apparently been cured. This may be a factor in an infection of this sinus when a history of an acute infection is lacking.

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*Read before the American Laryngological, Rhinological and Otolaryngological Society, Atlantic City, May 26-28, 1927.

Editor's Note: This paper received in The Laryngoscope Office and accepted for publication July 21, 1927.

Taking up the various symptoms of an acute infection, let us first consider:

Pain: This phenomena may be divided into headache and reflex or toxic manifestations. Headache, as Skillern has well said, "depends largely upon the pathologic condition present within the sinus". The pressure, whether the result of congested and swollen membrane impinging on its fellow membrane of the other walls of the cavity or through a thickened, tenacious pus filling the sinus and occluding the ostium, is likely to give symptoms of similar nature. A third cause or form of headache, the so-called vacuum headache or negative pressure, is considered unimportant and yet my experience shows many cases where this condition must have existed, as the end-results testify.

It may be true that it is an impossibility to have a negative pressure or closed cavity without the mucous membrane of the special sinus being inflamed, but this does not prevent the mucous membrane of the sphenoethmoidal fissure or more often a postethmoidal cell through swollen tissue or polyp formation occluding the ostium of the sphenoid from outside.

The headache is mainly situated in the occipital region, radiating in fan formation upward toward the vertex. Reflex pain with toxemic symptoms may extend over the temporal or mastoid regions; this is frequently accompanied with considerable muscular stiffness in the neck muscles and may even extend downward toward the clavicular region or as far as the humerus, scapular clavicular articulation. Two reflex pains, which I think are new and have never been associated with this condition, are, first, a reflex pain felt over the canine tooth of the side affected. Second, pain of a burning or continuous stab-like character felt at the junction of the soft palate with the anterior pillar of the faucial tonsil.

Pain back of the eye or eyes: This is usually dull in character and assumes a feeling of pressure exerted from behind forward. This symptom or pain is not increased by pressure made over the eyeball or lid.

One of the most frequent, and we might say prodromal, symptoms of sphenoidal infection is scintillating scotoma. This may take the form of bright spots in the visual field or these spots may be black, frequently taking the form of feathers which flicker through the vision.

Vertigo: Another symptom which is usually present either in a continuous or interruptive nature. This symptom can be present whether the patient is in the recumbent or the upright position, and

may be so pronounced and distressing in character, coming on in the middle of the night, that a firm hold of either side of the bed may be necessary to prevent falling out. Should a bilateral involvement of virulent toxemia be our problem, the vertigo may be characterized by a rotary as well as lateral motion. Stooping forward or rapid change or turning of the head will produce symptoms from which the patient has considerable difficulty to prevent falling. A peculiarity of gait as well as direction is noticeable in some cases. This is brought about by a sensation while walking of the buildings leaning toward the center of the street, and those having this symptom walk toward the curb, and have considerable difficulty in keeping to a straight line.

Sleeplessness: In spite of the general toxemia present, the majority of sufferers are unable to sleep with any degree of comfort. Frequent wakenings render their night miserable. A feeling of something breaking or discharging into the throat periodically is typical of the manner in which the sphenoid discharges. The swollen membrane of the ostium acting as a valve until the pressure within becomes sufficiently great to force an opening, when a certain amount of pus is discharged, the membrane closing again, to be forced open when the pressure is again exerted.

Malaise and forgetfulness, with inability to concentrate or take interest in their surroundings are symptoms usually elicited. These are the subjective symptoms we have found associated with acute infection of this sinus. The objective symptoms are few if any, and mainly consist of congested postethmoidal mucous membrane and swollen tissue in the sphenoethmoidal fissure, with possibly a discharge discernible in the olfactory fissure and on the pharyngeal wall, or we may see the discharge coming from the sphenoid ostium. A temperature of 99½ to 101½° is usual for this infection.

CHRONIC SPHENOIDAL INFECTION.

Perhaps no other sinus presents such a wide deviation in the subjective, objective and toxic symptoms as the chronically diseased sphenoid. I have divided these various symptoms into several groups.

Frist, the objective: Should a secretion be present, it is either seen coming direct from the ostium or as a tenacious discharge of a dark yellow or greenish color clinging on the pharyngeal wall. This discharge quickly changes into scale or scab formation and may by capillary attraction invade the olfactory fissure, as well as the choana. The typical cacosmic odor is perceptible on the breath, as well as from the nasal chambers. Enlargement of the blind spot is also a

condition to look for. Paralysis of the external ocular muscles or muscle may occur.

A. The mucous membrane when cleansed will be found to be hyperplastic in nature.

Second, the subjective symptoms will be divided into:

A. Pressure symptoms. *B.* Mental symptoms. *C.* Ocular symptoms. *D.* Toxic symptoms. *E.* Olfactory disturbances. *F.* General systemic disturbances. *G.* Complications.

Pressure symptoms radiating in various directions may be due to, first, pressure of the secretion or swollen membrane within the cavity, or, second, to mechanical pressure from the actual edema of the parts from venous stasis, or, third, to the cavity containing benign or malignant growths, as polyps, angioma, etc.

Pain from symptoms of the first and second groups occur periodically, lasting from one to several hours, but do not leave entirely. Pain from the third group give rise to a continuous symptom of pain, which is not relieved by position or ordinary medicinal treatment. Of these pains, headache radiating in the same manner as mentioned in the acute variety is present in the majority of cases.

Pain back of the eye is more constant and pronounced. The character of these pains depends entirely upon the pathological condition encountered, and may be described as dull, throbbing or semithrobbing, or combined. It may be diffused, not corresponding to the area of distribution of any special nerve. Any physical exertion, such as walking and running, or the use of tobacco or alcohol, will increase this symptom.

Mental excitement seemingly adds oil to the fire. Cohabitation is practically impossible, due to the excessive mental excitement and the depressed general condition.

B. Mental symptoms: These and the *D. Toxic symptoms* should be considered together, as it is due to toxemia we have the disturbances in loss of memory, dullness of faculty, general depression, malaise, inability to concentrate, aversion to work and lack of initiative, etc., whereas the toxic symptoms usually considered as those occurring at distal points. Of these, vertigo with stiffness of the neck muscles, both at the sides and back, are probably the most frequent. Those producing pain over the canine tooth, palate, glossopharyngeus muscle, in the muscles of the leg, over the instep (simulating the pain of flat foot), tinnitus aurium, with dimunition of hearing, are less frequent, although any one or a combination of them may be the only diagnostic factor of our case.

C. Ocular symptoms: Frequently conditions occurring in the eye or vision are the causative factor for patients to be referred to the rhinologist or sinuologist. Two cases, the report of which is part of this paper, were referred to us on account of sudden blindness in one eye; all other symptoms usually associated with this infection being absent. Examination, however, revealed a chronic sphenoid infection with an apparent acute exacerbation of their old condition. These two and one other case of similar nature have come under our observation in the past three years in which acute blindness has been the only symptom elicited from the patient.

Sudden unilateral blindness may be due to compression of the optic nerve in the optic foramen or by perineuritis or toxemia.

Exophthalmos, with or without edema of the conjunctiva, may be present to a small or large degree. Many of our cases show the eyeball completely dislodged from its socket.

Scintillating acotoma and diplopia are frequent manifestations. In one of our cases, polyopia of seven images was the outstanding feature. Lacrymation, photophobia and

E. Olfactory disturbances: Due to the mucous membrane in the olfactory fissure and that covering the septal side of the middle turbinate and septum being congested and frequently covered with pus. Olfaction, or the sense of smell, is diminished or lost entirely.

F. General symptoms: Sore throat, laryngeal cough, aphonia, hoarseness, bronchitis, gastric disturbances, such as loss of appetite, tendency to vomiting; this may be due to gastrointestinal disorders, or a hacking and reeching to dislodge from the nasal pharynx the tenacious secretion and adherent crusts.

Hepatic and gall bladder infection with a new symptom, a yellow colored soft palate, is frequently met with. Pain over the instep is also a new symptom to be associated with this disease.

C. Complications and sequela: Thrombosis of the cavernous sinus, due to the intimate anatomical relation between these structures, the cavernous sinus may become readily infected. Retrobulbar neuritis, with sequela of partial or complete loss of vision, is a complication too frequently met. Basal meningitis is observed more frequently than thrombosis of the cavernous sinus and I have never seen a case, whatever the treatment may have been, get well. Occasionally we meet with a case which has been treated surgically, a large opening in the anterior wall, with free drainage being made, and yet our symptoms, or some of them, return in a short time, following their complete disappearance. Examination by the pharyngoscope reveals the opening closed either by the mucous membrane acting as a cur-

tain, or the former sphenoid cavity partially or entirely filled with granulations. Pus or discharge oozing from the sinus or tissue within.

NONAERIATION OR NEGATIVE PRESSURE TYPE OF SPHENOID SINUSITIS.

It has been my experience that this type of infection or condition of the sphenoid is the most virulent of all conditions met. How many before we have from the history of the case or from some of the foregoing symptoms, opened the sphenoid sinuses to find apparently a normal condition present. And yet, within a few hours, all or most of the patient's symptoms subside or leave. Can we explain this? No. Should we say that due to the absorption of the oxygen normally in the sinus, which is now absorbed by the circulation, that the resultant negative pressure will give the virulent toxic symptoms? Personally, I am unable to believe it. And so we are brought to a position of the hypothetical question, as our friends the lawyers would say. It is a well known fact that the nonaerobic germs are the most virulent, giving symptoms and being productive of conditions of the most pronounced, patients seldom recovering from their invasion. It's not reasonably sane to advance the thought that in the nonaeriation or so-called vacuum variety of sphenoid infection, that a nonaerobic infection may be the cause and by operative means aeriation takes the place of the formally closed cavity with the elevation of the toxemia and other symptoms.

So many cases of sphenoidal infections present themselves to our clinics, after having "gone the rounds" without relief, that one is reminded of Professor Parvin's maxim of "Be careful or you will find what you are looking for".

The following case reports will illustrate some of the symptoms already considered.

Case 1: H. J., male, age 48 years, merchant, was referred on Sept. 23, 1926, by Dr. J. M. Anders for examination, to try and elicit a cause for his sudden loss of vision in the right eye.

Nasal examination revealed: Right nose, several polyps in the ethmoidal region partially occluding the middle nasal pasage. Considerable pus in this nose in middle nasal passage and olfactory fissure. Mucous membrane hyperplastic and congested.

Left nose, passages and mucous membrane normal, except on floor and part of inferior turbinate, a slight amount of pus coming or blown from the pharynx or right nose into this side.

History from patient and only subjective symptoms given, is sudden blindness in right eye.

Sept. 20, 1926: Temperature 99°; urine, S. G. 1.015, reaction alk., sugar neg., faint trace albumen, minute shreads, no casts; W. B. C. number; blood coagulation 4 minutes. Diagnosis: Sphenoid sinusitis. Treatment: Sphenoidotomy. Result: Vision gradually returned to normal.

Case 2: D. M., male, age 32 years, salesman, referred by Dr. Horwitz on Dec. 17, 1926.

Nasal examination negative as to any pathology. Septum slightly deviated superiorly to the left. History from patient. Has never been seriously ill. No attacks of influenza. Seldom catches cold. Complains of more or less constant pain at junction of soft palate and anterior pillars on left side, pain is burning in character. Position of the patient seems to have no effect as to the severity of the pain. Has had this distressing symptom for past four years. In consultation with Dr. Ross Hall Skillern, we decided to open the left sphenoid sinus. Temperature had been subnormal, 97.3° to 98°, for past year. Sphenoidotomy was done with entire relief of his pain and the temperature became normal in three days following the operation.

Case 3: A. H. C., male, age 67 years, farmer. Symptoms for past five years: has had excruciating pain over the pyriform crest on the right side of face just above the canine tooth socket. This is continuous, usually dull in character. Occasionally a sharp lancinating pain is felt for a brief period. Pain continuously in calf of right leg, which frequently incapacitates him from performing any of his usual duties. Has never had any severe illness and is a well developed and preserved man. Through recommendation and suggestion from his family physician he has had all of his teeth extracted, without relief. X-ray is suggestive of sphenoidal involvement, although no visual findings suggest this. Examination of the nose is negative as to pathology; neither can I elicit any further subjective symptoms.

On Jan. 11, 1927, under cocain anesthesia, the sphenoid sinus was opened, with almost immediate relief of his pain, both in the face and legs.

His temperature on admission was 99° and on the fourth day following his operation became normal, where it stayed for the two weeks I had him under observation.

Urine and blood both negative as to any connection with his symptoms.

Case 4: H. H., female, age 55 years, houseworker. Was referred to me for acute blindness in right eye by Dr. Luther Peters. The

condition of blindness developed suddenly on the morning of Jan. 11, 1927. She had a severe attack of influenza three weeks prior.

Examination of both nares negative; no discharge on pharyngeal wall. I was unable to elicit any of the generally recognized symptoms associated with sphenoidal empyema, no headaches nor vertigo. Her history seemed to begin and end with the one condition, acute blindness.

On Jan. 20, a sphenoidectomy was performed on the right side. The interior of the sphenoid cavity appeared normal in color, neither was there any discharge.

On Jan. 21, Dr. Peters' report of the eye findings were: Pallor of fundus. Veins are fuller than normal and the arteries smaller; several are seen indistinctively and show a very tiny stream.

On Jan. 24, edges of disc show a well defined elevation. Vision is improving and now can distinguish hand movements.

On Jan. 25, patient is able to count fingers at 14 inches.

On Jan. 31, fundus clearer, slightly pale to nasal side. Retina shows a small white area.

The temperature was normal throughout her stay in the hospital. Urine: S. G. 1027, acid, albumin and sugar negative. No casts. Blood count at time of operation: Hb. 82, R. B. C. 4,280,000, W. B. C. 7,750, P. M. N. 79, S. L. 17, L. L. 2. The Wassermann reaction was negative. The blood chemistry gave the following: Sugar, 91.7 mgn./100 c.c.; N. P. N., 35.3 mgn./100 c.c.; Creatine, 1.47 mgn./100 c.c.; Uric Acid, 2.7 mgn./100 c.c. Blood pressure was 116 over 66.

Case 5: K. McA., female, age 51 years, married, houseworker, gave the following history: Since Christmas, 1926, has had occipital pain or headache; these attacks were paroxysmal, coming on at different hours, both during the night and day. She was frequently disturbed and awakened at night. Pain on occasions would radiate to the shoulder, neck and mastoid region, cheek, or, on several occasions, would be the apparent cause of nausea and vomiting. The discharge would always be in the back of the throat and would be intermittent in character. Had several attacks of scintillating scatoma in the form of feathers, which would last four hours, to disappear, only to reappear in a few hours or on the following day. Suffered with severe pain back of the eyes and also at the juncture of the soft palate with the anterior pillar. Had arthritic attacks affecting both knees; a constant desire to clear the throat.

Diagnosis: Sphenoid sinusitis, right.

On Feb. 2, 1927, we opened the sphenoid, which was found decidedly diseased. She was kept under our observation for two weeks, when she returned home greatly relieved. On March 4, one month after her operation, she returned in a condition almost the same as when we first examined her, the symptoms all having returned. On examination a hyperplasia of the mucous membrane, with a general formation of granulations had taken place within the sinus cavity and pus could be seen oozing from between the flaps.

Again we cut and curetted away all tissue within the sinus, with relief from her symptoms, and she returned home.

On March 6, examination again revealed a similar picture as before stated and an X-ray examination with probe in situ confirms our diagnosis of the cavity being practically filled with granulation tissue. She is coming for another operation, when we are hoping for better results.

Case 6: M. H., female, married, age 60 years, houseworker. This patient, the wife of a physiologist, was referred to us to find the reason for her vertigo. The ears had been most thoroughly examined by an otologist, with negative findings.

She gave the following history: On Aug. 16, 1926, she had a severe attack of influenza, necessitating her remaining in bed for three weeks. Following her apparent recovery from this influenza, she developed a vertigo which was continuous and of decided severity, sufficient to totally incapacitate her from performing any of her usual household duties. This condition gradually became so severe that she was compelled to keep to her bed, and even in this recumbent position a nurse was essential to minister to her care. The only other symptoms we were able to elicit was pain about the size of a French pea over the canine tooth on either side. Examination by the nasal pharyngoscope revealed a small stream of mucopurulent discharge coming from either sphenoid sinus.

On Feb. 14, 1927, the right sphenoid was opened in the usual internasal manner by the Farachi forceps and a few days later the left sphenoid was operated on by the same method. The pain over the canine teeth was immediately relieved at the time of the operation. The vertigo gradually disappeared.

18th and Chestnut Streets.

A NEW METHOD FOR SKINGRAFTING THE MASTOID CAVITY. A NEW METHOD FOR CLOSING OFF THE EUSTACHIAN TUBE AT ITS TYMPANIC END IN THE RADICAL MASTOID OPERATION.

DR. ALFRED KAHN, New York.

Perhaps the prime object of the radical mastoid operation is the cure of discharging ears. The operation is usually indicated after all other measures have failed. An otologist of a certain school of opinion may maintain that the radical mastoid should only be employed where there is dead bone in the tympanic cavity or in the walls surrounding it; in other words, local death of bone keeping up a constant irritation and hence a constant discharge. In discussion this is how the surgeon may view the radical mastoid operation. A patient, however, knows nothing of necrotic bone. He comes and consults because he has a discharging ear. There may not be necrosis, the discharge coming through the external auditory canal may come through a large perforation in the drum, through the tympanic cavity and Eustachian tube from the throat. He wants a cure. The otologist has tried every therapeutic means and failed. The radical mastoid operation is the only means left at his command. The patient consents and the radical is done. The local area of necrosis may be removed. A most excellent radical mastoid has been performed. The wound heals, but the discharge continues. In nearly every instance through the Eustachian tube, middle ear, out through the external auditory canal; and the patient is not satisfied. What he wants is a dry ear. That's what he came for. He doesn't consider the operation a success, and the surgeon can't convince him otherwise. There is disappointment on all sides.

The Eustachian tube is the bugbear. It is almost impossible to close it off. Hence there is a constant discharge up through it from the throat. All kinds of schemes have been devised to close it off. The most successful of these various procedures is a skingraft. A Thierch graft is cut from the thigh and placed in the tympanic cavity over the upper opening of the Eustachian tube. The chances are that in over 50 per cent of cases operated the graft doesn't take; and even though it may take, it almost invariably fails over the opening

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of the tube, and the discharge continues. Now, why does the graft fail to take? The graft must, once it is placed in the mastoid cavity, immediately make contact with the bone over which it is placed to absorb sufficient nourishment to permit it to survive. Therefore what you really have are loose cells cut off from their blood supply, lying loose; trying to absorb nourishment from hard bone. To my mind, even though the graft takes for the time being, in many instances the skin comes away weeks, months or a year or more, later. The graft is never sure to have a firm bed. The surgeon may succeed by means of his Thierch graft in getting a quickly healed wound in

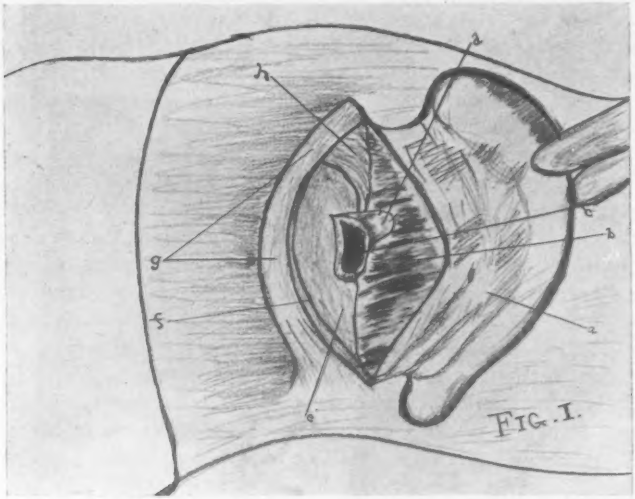

Fig. 1. Diagram showing radical mastoid cavity. Incisions made outlining Kahn's skin flap. "a" Auricle turned forward. "f" Line of mastoid incision. "b" Anterior flap of mastoid wound turned forward. "c" Mastoid cavity. "d" Usual radical mastoid flap made from the canal. "g" Kahn flap to be dissected up and the pedicle turned forward into the mastoid cavity. "e" Mastoid bone. "h" Temporal muscle.

a minimum number of cases, but it is only by the merest luck that he closes off the Eustachian tube. Up to this time there is no sure method of closing off the tube.

I have devised a technique different from any scheme yet tried. It permits of a continuous blood supply to the graft. It is only necessary for the graft to attach itself to the underlying bone and it is practically sure of doing this. It is almost impossible for the graft not to take. The technique is as follows: The radical mastoid is proceeded with in the usual way until it is completed, then an incision is made in the skin following the original incision which first exposed

the mastoid bone. This incision is made from above downward and is from a quarter of an inch to an inch back of the original mastoid incision, depending upon the width of graft desired. By means of a pair of scissors the upper edge of the original mastoid incision and the upper edge of this new incision, a quarter of an inch to an inch back, are united at their upper ends, thereby creating a pedicle flap (Fig. 1 g). This flap is turned forward on its pedicle and is made to line the tympanic cavity and mastoid cavity, and in this process is made to cover the upper opening of the Eustachian tube. G and G1, Fig. 5, shows the flap mounting through the mastoid cavity, over the

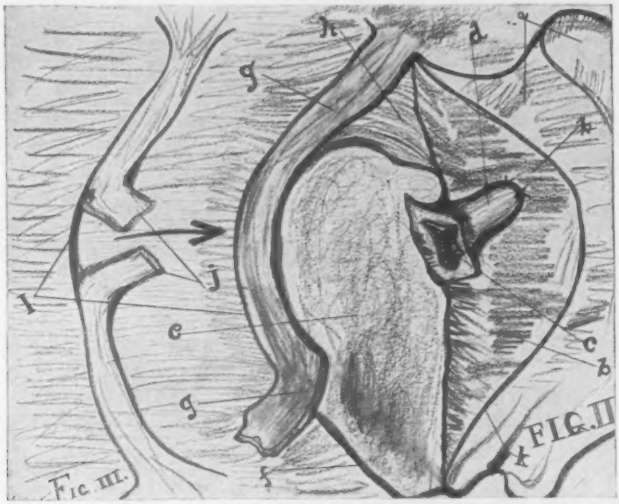

Fig. 2. Diagram showing radical mastoid cavity. Kahn's flap is dissected from below, showing the base of pedicle of the flap above. "a" Auricle turned forward. "b" Anterior flap of mastoid wound. "c" Radical mastoid cavity. "e" Mastoid bone. "f" Line of mastoid wound after Kahn flap is dissected up. "g" Kahn flap partially dissected from below. The base of flap is above. "h" Temporal muscle. "l" Extension of "f" and incision outlining Kahn flap. Kahn flap is thrown forward into the mastoid cavity, and the mastoid wound is closed by uniting "k" to "f".

Fig. 3. Diagram showing how Kahn flap can be cut in half, the upper half of the flap receiving its blood supply from above; the lower half of the flap receiving its supply of blood from below. "j" Ends of cut flap to be used as lining for mastoid cavity. "l" Line of mastoid incision after flap is dissected up.

facial ridge into the tympanic cavity which it lines, over the Eustachian tube, which it plugs firmly. Fig. 1 g shows the flap with the base of its pedicle downward. Fig. 2 shows the flap with its pedicle base upward. Fig. 3 j shows the flap divided, making two pedicles. In this way the flap can be altered in various ways. The flap is packed firmly against the walls of the mastoid and tympanic cavities

by means of gauze, which should be left for several days, until the flap attaches firmly.

The mastoid wound is then closed as follows: I have not gone into the detail of taking flaps from the anterior edge of the mastoid wound, but this can be done, using the same procedure described for taking the pedicle from the back flap of the mastoid wound. I will assume that the flap is taken from the posterior flap of the mastoid wound. After the pedicle is packed firmly into the mastoid cavity

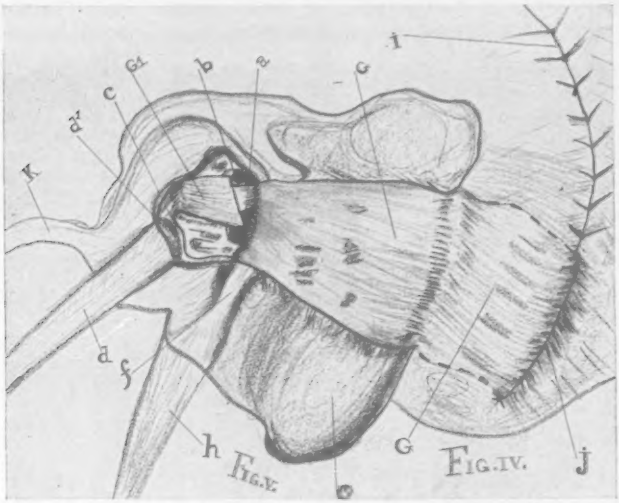

Fig. 4. Diagram showing method of closing the mastoid wound over the skin flap. "i" The mastoid wound is closed above by wide apart ligatures. Below "j" where Kahn's flap tucks under the anterior flap of the mastoid wound, the wound is closed by very thin catgut ligatures, as described in the article.

Fig. 5. Diagram showing Kahn flap passing over mastoid cavity; over facial ridge; into and lining middle ear and covering the upper opening of the Eustachian tube. "e" Mastoid. "f" Facial ridge. "G" Kahn flap passing over mastoid cavity, over facial ridge, into the tympanum. "G1" Kahn flap lining middle ear ,and covering Eustachian tube "d1". "c" Middle ear. "a" Round window. "b" Oval window. "d" Eustachian tube. "h" styloid process. "k" Zygomatic process.

the anterior flap of the wound is brought back to be stitched or clamped to the posterior edge of the wound. This is after the pedicle has been taken from the po terior flap. This is illustrated in Fig. 4 i. The lower part of the wound, assuming that the base of the pedicle of the flap is down, is clo ed by very thin ca gut sutures. The pedicle passes into the mastoid cavity under the anterior flap of the mastoid wound. This tucking under of the pedicle brings the edge of the anterior flap of the wound in contact with the base of the pedicle flap. An extremely light incision is now made across the pedicle flap

(from above downward). The pedicle flap is now sutured, along the line of this incision, to the anterior flap of the mastoid wound. This suturing is done as mentioned before with very thin catgut. This last procedure has the effect of closing the lower angle of the mastoid wound and is illustrated in Fig. 4 j. The whole mastoid wound is now closed and drainage takes place as usual through the external auditory canal.

Recapitulating, the mastoid cavity and tympanum are now lined with skin, that skin having a constant blood supply.

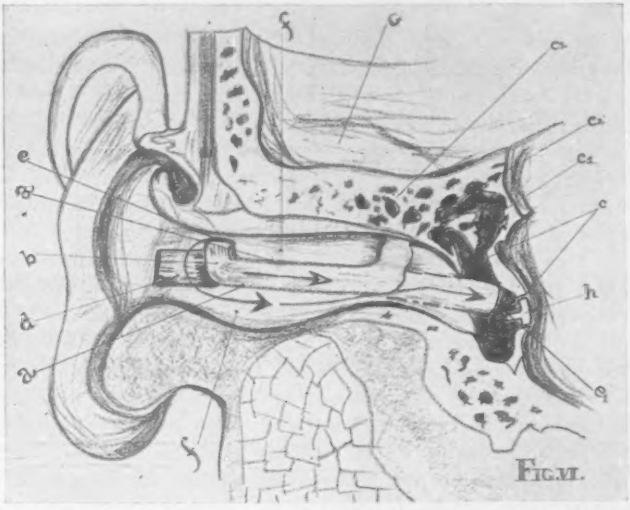

Fig. 6. Diagram showing the technique of taking a pedicle flap from the external auditory canal. "f" External auditory meatus. "c" Middle ear. "h" Stapes. "C1" Epitympanic recess. "C2" Mastoid cells. "G" Floor of the middle fossae of the skull. "a" Upper and lower lines of incisions outlining flap. "d" The flap is dissected up from its floor so that the point "e" is turned on its pedicle to cover any point in the middle ear that is desired, say for example over the upper opening of the Eustachian tube, say to the point "e1". "b" The flap. The flap is turned toward the tympanic cavity as indicated by the arrows.

Method of taking a graft from the external auditory canal: A pedicle graft can in the same way be taken from the external auditory canal. The procedure is illustrated in Fig. 6, which is a longitudinal section through the external auditory canal. The pedicle is best taken from the floor of the canal. The flap is outlined by an upper and lower incision which is joined at its outer end, the base of the pedicle being toward the middle ear. The pedicle is turned on its base and transplanted at the point in the middle ear desired.

48 East 49th Street.

ARSENIC TREATMENT OF DEAFNESS.

DR. E. WODAK, Prague, Czechoslovakia.

The interest with which my papers published in the Muenchner Med. Woch., 1925, No. 28, and the Deutche Med. Woch., 1926, experience with the treatment under consideration in an American No. 13, respectively, were received prompts me to present my new scientific journal.

At the time of writing, the number of cases treated by this new method much exceeds 300, and though no substantial variations from my earlier results can be recorded, some points have been cleared up.

The new method of treatment has remained unchanged, but it is not schematic. It should rather be adapted to the individual case. I start with one pill of 5 m.g. Natr. arsenic, to be taken during one meal, preferably in the evening, and I watch the reaction of the patient to it. This cautious manner is recommended since one must always reckon with an arsenic idiosyncrasy. If there are no bad effects, I give two doses the next day, to be taken one at noon and one evening, and the following day three, one with each meal. In slight, and less severe cases of deafness, I do not change the dosage; in more severe cases, I increase the dose to four pills daily. If symptoms of indigestion or intestinal trouble develop, I stop the treatment immediately, and wait until these symptoms have entirely disappeared. Then I begin again with one pill, and slowly increase the dose. Occasionally the arsenic is not tolerated at all. In such instances, I administer Natr. arsenic hypodermically. I start the patient with 2 m.g. and gradually increase to 20 m.g In this latter method symptoms of indigestion and internal trouble are felt by some cases, too, but in a certain percentage of cases where arsenic per os does not agree with the patient, treatment may be given hypodermically.

Toleration of the arsenic and reaction to it of the patient having been ascertained, the treatment should be continued until 40 to 60 pills, or 200-300 m.g. Natr. arsenic have been taken, according to the seriousness of the case. Then the treatment must be stopped gradually, and the patient given a complete rest from three to four months. This interval is necessary because the system does not rid itself of arsenic completely before that length of time.

Up to now, apart from the passing symptoms of indigestion, or intestinal complaints mentioned, I have failed to find any serious

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disturbances. Even where complaints of general restlessness and headache, etc., have made themselves manifest in a few cases, these have quickly disappeared when treatment was discontinued.

Cases That May Be Treated By Arsenic: The arsenic treatment may be applied to affections of the middle ear, as well as cases with inner ear trouble. In both instances about the same percentage of success has been recorded. The less advanced the case, the better the chance for success. Cases where hearing has been reduced to colloquial talk to 10-inch, little result may be expected. This does not mean that such cases ought to be excluded from the start, but it may serve as a warning not to promise the patient too much.

Cases in which hearing improves rapidly immediately after the first dose of arsenic give the best prognosis. With these the improvement lasts longest, too. Of course, this applies to moderate cases of deafness, whisper tones, 3-6-inch.

Cases of deafness of the highest degree very rarely show such improvement. Even the most that one may expect even in cases that yield best to treatment is to increase hearing gradually 200-300 per cent, and even this improvement is prone to disappear rapidly. Still, even such partial success with advanced stages of deafness is valuable, since the patient, on the verge of despair, skeptical towards all therapy, appreciates even the smallest improvement. However, the treatment ought not to be tried on hopeless, or very far advanced cases, lest the poor results obtained with such patients discredit the whole method.

Since it is very difficult to distinguish between spontaneous improvement in hearing and improvement due to the arsenic treatment, all such doubtful cases I have recorded in my summary as doubtful, even though I had the firm conviction that the beneficial change had been brought about by the arsenic. There also were cases where hearing was increased 50-100 per cent. But since this improvement occurred with patients who were practically deaf, I have not placed them among the successful cases, although they might so have been considered theoretically. I have put all these and similar cases in the negative column.

CASES GROUPED ACCORDING TO THEIR ETIOLOGY.

1. Otosclerosis, 56 cases: 13 no effect; 9 doubtful. Total, 22 cases negative (39 per cent), 34 full success; 11 times improvement of hearing up to 100 per cent, 14 times from 100-500 per cent, 7 times till 1000 per cent; 2 cases showed 30 times increase of hearing. Among these latter, one case went up from 1′ to 27′ and remained at this pitch during the time of observation (two months).

2. Chronic middle ear catarrh, adhesive processes, etc., 74 cases: 24, no effect (32.5 per cent); 50, positive improvement; among them with 31 with more than 100 per cent increase in hearing.

3. Neuritis cochlearis, 24 cases: 58.5 per cent positive success.

4. Presbyacusis, 10 cases: 3, no results. The number in this group and other groups (lues) is too small to allow reliable computation. The advanced age of the patient is no hinderance in the treatment. I have used it successfully with people age 70 years.

All together, I have treated 213 auditory organs: 82 with no result, 131 (62 per cent) successfully.

The action of the arsenic may be explained briefly. Originally, we believed that the vessels of the inner ear were influenced by the typical action of th arsenic, palsy of the capillaries, with improved circulation and therefore better functioning. But since arsenic shows a certain effect in nearly all types of deafness, it may also cause a greater sensibility of certain parts of the cortex of the brain or nuclei. One must consider, too, the possibility of an arsenic neuritis. Such an explanation could perhaps be considered in those cases where we find an improvement in the hearing only after several weeks of treatment, very often not until the completion of it. It is also possible that in some cases several causes mentioned above are effective at the same time; at any rate, the theoretical side of the arsenic treatment of deafness is not yet quite clear, and requires further investigation. The purpose of this paper is to draw attention to this kind of treatment and have it further tested.

Since the publication of my earlier statistics, the number of cases treated have very much increased. But I could only note in the present writing those cases which have been under my observation a long time.

The number of negative cases compared with those in my earlier publication has increased from 34 per cent to 39 per cent. I suppose this is partly due to the fact that after my method became known I received a large number of hopeless cases for treatment; cases in which success was impossible. This explains the poorer showing of my more recent statistics.

INTRACRANIAL COMPLICATIONS OF OTITIC ORIGIN WITH REFERENCE TO DIAGNOSIS AND MANAGEMENT.*

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In the consideration of these cases, both from the standpoint of diagnosis and treatment, we must organize our meager stock of information from whatever source possible and fortify against a tremendously disorganized and varying group of symptoms and signs, and after a conscientious consideration of all data at hand, trust and hope that our judgment was correct.

There is not a group of cases in the domain of surgery wherein the patient's condition is more serious, and the evidence upon which operation must be undertaken more meager, nor the effects of mistaken surgery more disastrous.

An abdominal case, if uncertain, one may nearly always explore with a reasonable amount of safety, while in a head case the results of an exploration can never be foretold, and in the event of a negative finding one is left as much in the dark as before.

Besides the grave responsibilities of the suregon to the patient, his relation to the patient's family is not less unpleasant. Having often to make his decision to operate upon uncertainties certainly does not lessen the weight of the responsibility, and his liability to blame in the event of an unfavorable termination is markedly increased. A personal element, therefore, forces itself upon us for consideration.

However, we have to make the best of the situation.

In speaking of intracranial complications of otitic origin I have not limited myself by minor differences, as one might find in a textbook as, for instance, that labyrinthitis is not really an intracranial condition. Nor is it my intention to give a classic description, and in a certain order, of those conditions as they are of necessity described in a textbook. First of all, I am not writing a textbook and, secondly, a sick person does not usually follow textbook descriptions nor instructions. I wish merely to state briefly the problems as they come to our attention at the bedside and give some little consideration to their solution.

When I speak of intracranial complications I refer to any trouble within the head that may arise as a result of an infected ear. These may be mentioned as extradural abscess, subdural abscess, sinus

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thrombosis, meningitis in its various manifestations, labyrinthitis, cerebral and cerebellar abscess. When anything goes wrong in a patient with an ear condition it is the safest procedure to think of them all and suspect any one or more of them.

There are two general types of patients who present themselves to an otologist which should be considered in the light of intracranial disease, and they are: First, and probably the less frequent, the cases of acute otitis media, with or without mastoid infection; and secondly, the cases of chronic purulent otitis media. The last-named cases fall into two divisions: *a.* the class of cases with a frank chronic discharge, which may at any time without warning light up into a serious intracranial condition; and *b.* the cases of an occasionally recurrent mild type of otorrhea, which is often forgotten by the patient himself. It is this mildness which makes them all the more serious as they are as troublesome as the other cases and the physician must think of it in order to extract this information from the patient when the latter's illness seems suspicious.

When we consider the cases of acute purulent otitis media in the light of intracranial complications we have at least one factor which may be somewhat comforting and that is that such cases are rather infrequent, forming but a small percentage of the total number of intracranial conditions. This fact should occasionally act as a restraining influence when an intracranial exploration is considered and may needlessly be done.

Acute otitis media has its usual course and duration and it may also have its variations. However, the great majority of cases terminate in recovery. Its most common compliction is, of course, mastoiditis. Occasionally in infants, a primary jugular bulb thrombosis may arise. Some consideration will be given to the latter condition in its proper place.

When mastoiditis arises, that too has its tendency to recover, sometimes with operation and often without. When the condition becomes bad enough for operative interference the prognosis is also good in most cases under proper management. When, however, something goes wrong during the postoperative period then we become worried, chiefly about intracranial complications. Ordinarily we expect some temperature elevation, the pulse and respirations in accordance with the temperature. Perhaps a greater rise may be seen as a part of the postoperative reaction, but then a gradual decline to normal within a week or ten days. Other symptoms, like pain, restlessness, insomnia, etc., are expected to rapidly disappear.

When, however, neither the temperature nor the symptoms show any tendency toward amelioration, we may think of some intra-

cranial complication, but all other possible causes must be eliminated. Often a stitch abscess, a collection of pus within the wound, may explain everything. We must also not forget that a patient who has been operated on for mastoiditis is not immune to other ailments. The latter have been found in many forms. Tonsillitis, diphtheria, opposite ear involvement, pneumonia, with or without meningeal irritation, nephritis and even appendicitis, are conditions which I have seen develop in patients during their course of illness with mastoiditis. These and similar conditions should be ruled out before we come to the point of deciding for an exploration of the head for intracranial complications.

In chronic purulent otitis media a somewhat different circumstance exists. Here we have to be pushed, before we dare diagnose or even wake up to the suspicion of intracranial disease. This is particularly true in those cases of chronic purulent otitis media with very little or an occasionally recurrent moisture in the ear. The patient becomes sick suddenly and may be treated for almost any condition before intracranial disease is even given consideration. For instance, one case was treated for gastric disturbance, having suffered from nausea and vomiting and other gastric distress, with slight dizziness, and because after a few days the dizziness became worse the medical attendant asked me to see the ears, which were chronically discharging. When I examined the patient I found a definite labyrinthitis.

Another case of mine was that of a man who sought medical treatment and was admitted to a hospital and carried through a course of pneumonia, from which he definitely suffered. He had a chronic purulent otitis media, which annoyed him off and on, but he did not call his medical attendant's attention to it until he had severe pain about the ear. But at that time he already had a sinus thrombosis, a large temporosphenoidal abscess, and a large cortical abscess in the region of the parietal lobe. I am not certain, but I have a feeling that his pneumonia was secondary to his sinus thrombosis, and his brain abscess secondary to his sinus condition or his pneumonia. The severity of the latter forced him to seek medical aid while, because the ear did not annoy him any more than usual, he took for granted that it must be so, and paid no attention to it. It would seem that it is imperative for any physician who is called upon to treat patients with acute ailments, especially when the diagnosis is somewhat doubtful, to have the ear in mind during the examination. When the history or presence of a chronic ear infection is elicited all efforts should be bent towards its elimination from guilt before a definite diagnosis of another ailment is made.

Now, after all is said and done, in the case of acute otitis media all factors which may explain the continued illness of the patient have been considered and eliminated; in the chronic case no correct explanation nor definite diagnoses of the patients' ailments have been made, what are the circumstances which should focus our attention to the question of intracranial disease?

There are a few symptoms which may be present in combination and even more often any one alone which may compel our attention to the head. The first and foremost is headache, which may be severe or, if not severe, it may be persistent. Often it may be both severe and persistent, but at any rate headache points to some trouble in the head. Second in importance is the symptom of vomiting, its occurrence with headache, or its persistense alone in spite of no previous gastric ailments points to the head. Vertigo is another head symptom. Fever, chills and a disturbed ratio between the pulse, fever and respirations is suggestive of head involvement. Of course, when there are localizing neurological signs there is no more question about the existence of an intracranial condition.

However, these symptoms, with the exception of localizing signs, may be symptoms of almost any other ailment. Acute indigestion or influenza may have all of them. It is only after proper evaluation of these symptoms as to their actual character and order of occurrence in the presence of a probable etiology in the form of a discharging ear that they become of value. Before these symptoms may be properly placed and their value clearly estimated, some facts about intracranial pathology which form the basis of these symptoms must be carried in mind.

Let us visualize an otitis media with or without mastoid involvement and follow its probable activity into the cranium. In the acute form there is present an infection with inflammatory reactions. The tissues are in normal condition to resist the infection. The mucous membrane has a good blood supply, the bone which is healthy can partake of this blood supply and we can expect that unless the infection is unusually severe or that the individual's resistance is much below par that the infection will be limited to the middle ear or possibly the mastoid. The healthy state of the tissues and the average good resistance of the individual probably accounts for the lesser frequency of intracranial involvement in acute conditions. In the chronic otorrhea where there is an added infection in the form of an acute exacerbation, the situation is different. Here we have a devitalized mucous membrane with the fibrous tissue that had formed cutting off the blood supply from the underlying bone, diminishing

its power of resistance. Here is a vulnerable point for an active infection to make its entrance into the cranium.

Anatomically the direction in which disease can travel may be stated as follows: Upwards it may perforate the tegmen either of the middle ear or the antrum and so invade the meninges, where it may be successfully walled off and localized and only form an extradural abscess. If it is not walled off it may spread to the rest of the brain coverings and produce a generalized meningitis. If it is walled off it may still break its barrier upwards into the brain tissue and form a brain abscess. In the location of the tegmen the sphenotemporal lobe would be involved. If the invasion is backward in the mastoid the lateral sinus is first attacked. It may remain limited to the sinus region and its spread be limited by the very close adherence of the edges of the sinus to the cranial bone. In this way it would form a perisinus abscess. It may continue inward and break into the sinus, forming the various degrees of sinus thrombosis. It may still further continue inward and attack the cerebellum. The cerebellum may also be attacked through the dura, which is not directly concerned with the formation of the lateral sinus. In this case the process holds true with regards to the walling off and the probable continuation into the cerebellum, or in the absence of localizing power produce a generalized basal meningitis. Inwardly the infection may travel to the vestibule and produce either localized or generalized labyrinthitis, depending upon the severity of the infection and the localizing power of the tissues. Through the labyrinth it may continue by the way of an aqueduct beginning in the vestibule (aqueductus cochlearis), or the sheath of the eighth nerve into the posterior fossa or by direct necrosis of bone to either the posterior or middle fossa of the skull and produce meningitis.

A brain abscess may also be produced at some distance from the point of infection by the traveling of the infection along a blood vessel sheath and causing a thrombosis of this vessel, shutting off the blood supply to the part concerned. Then the tissues break down, causing an abscess. Such an abscess may tend to point through a narrow channel called a stalk at the roof of the middle ear or mastoid.

In view of what takes place what symptoms may we expect to occur as a result of infection? The meninges are the most sensitive and the most rebellious from the standpoint of symptoms, of all the tissues traversed. The sensory nerve supply is richest in the dura with its large vascular supply, therefore pain or headache is there most felt. The richness in blood supply of the dura and other coverings of the brain would favor absorption of toxins with result-

ant symptoms of constant absorption in the way of fever, pulse and respiratory changes, etc. So that in the presence of an etiologic factor like an otitis media when there is sudden onset of severe and persistent headache with fever, meningitis must be suspected. This symptom of headache is so constant that in its absence we are fairly safe in ruling out meningitis. Headache, therefore, severe and persistent, is the feature symptom of meningitis. Together with this we would expect to find symptoms which are a result of infection, like constant high fever, a chill or several chills and vomiting. Whether the infection of the meninges becomes generalized or localized the same is true, the difference being somewhat in the degree and particularly in the duration. Naturally there would be a lesser degree and a shorter duration when localized. If localization takes place and through surgical means, such as a mastoid operation, the condition is relieved, that is the end of it. If, however, after the occurrence of the symptoms mentioned with an apparent relief, temporarily, suspicious symptoms, of which we will presently speak, arise, we must infer that there has been an infection of the meninges which subsided perhaps by localization and instead of becoming a generalized meningitis had probably entered the brain substance and a brain abscess will likely result. These meningeal symptoms are spoken of as the initial stage of brain abscess. In making a diagnosis of brain abscess it should be borne in mind that those symptoms must have occurred and such information must be carefully elicited.

Thus far the chief symptoms that compelled us to think along these lines was headache. When fever is the outstanding symptom, our thoughts are led in a different direction, namely, that of the sinus, and while there may be some headache and a chill, all symptoms are held in abeyance while the fever is conspicuous. Furthermore, the fever is rather characteristic, it being caused by an entrance of toxic material into the circulation periodically, and eliminated periodically, the fever rises when the system is overwhelmed with the infection, and it falls when elimination takes place. It may fall to normal or almost so and is characteristically accompanied by sweats. These sweats are to be looked for as they are more or less indicative of efforts at elimination. When the sweats are absent we are justified in questioning the existence of sinus thrombosis. Another characteristic is often present and that is a feeling of well-being by the patient during the afebrile period. This may trap one into a sense of security which turns to disappointment with the next temperature rise.

When vertigo appears suddenly in an individual, with a probable focus of infection like an otitis media, our attention is at once directed to the labyrinth or cerebellum. When we can elicit a history of a chill, headache and fever which have subsided, or symptoms which point to sinus thrombosis we naturally blame the cerebellum, otherwise we make our studies in the direction of the labyrinth and attempt to eliminate it from blame.

So far I have spoken of these conditions in a more or less conglomerated fashion. The truth of the matter is that when we get a patient he comes to us with a conglomerated group of symptoms and only by discerning the important ones and classifying them, even though roughly, can we finally discover the road towards which our greatest efforts should be directed.

When, as we have mentioned before, fever is the most conspicuous symptom and headache is not particularly prominent, our attention is directed to the lateral sinus and we should follow this clue to the limit of our information. When the temperature range is characteristic, that is it goes up suddenly to about 103° F. or 104° F. and comes down again to about normal within twenty-four hours, accompanied by sweats, a chill, a high leucocytosis and a positive blood culture for the organism concerned in the primary lesion, or in the later stages metastases in the heart, kidneys, muscles, etc., our puzzle is practically solved and treatment is undertaken along the lines of sinus thrombosis. When, however, there are other complications present and the characteristic signs are wanting, the patient being quite sick, then we become more cautious about exposing our patient to the dangers of an elaborate operation, perhaps only to find that the patient's illness is due to some medical condition and that the sinus operation was needlessly done. On the other hand, we must bear in mind that a sinus thrombosis may exist and its presence be masked by some other coexisting condition. I think I could better illustrate my point by briefly referring to one of my puzzling cases of sinus thrombosis. The patient I refer to was a girl, age 11 years, who for three weeks suffered from an acute otitis media before she consulted her physician. Her eardrum had spontaneously ruptured. When I saw the patient she looked very toxic, she had a sort of greenish complexion, with a heavily coated tongue. There was some pain in her left ear and some tenderness over the mastoid region, as well as in her neck. X-ray showed merely some exudate in the mastoid, but no destruction of cells. Leucocytosis was high, about 20,000. From the toxic appearance of the patient and the tenderness in the neck I suspected that I might be dealing with a sinus thrombosis, the temperature also being high. The toxic appearance of the

patient is often helpful in making one suspicious of sinus thrombosis. The sickly pallor, or perhaps one may call it a lividity, perspiration and extremely coated tongue seems to be a picture more marked than is ordinarily seen in a simple mastoiditis. I recall having seen one boy with such a picture, who walked into the dispensary with some evidence of mastoid disease. I sent him over to the operating room for Dr. Coates to operate on the mastoid, which he did. At the operation a necrotic sinus was found. To return to my patient: while her toxic appearance looked suspicious there was no question that at least a mastoidectomy was strongly indicated. This was done and the surface cells seemed well intact, but the deeper cells were destroyed and there was considerable pus under pressure at the tip of the mastoid. The lateral sinus was purposely exposed for investigation and found to be normal in appearance. Gas anesthesia was used because of a cough. The temperature was high for a few days and made some excursions, but gradually came down to about normal and then went up again, made up and down excursions for about two days and then went up to 103° F. and remained there for about three or four days, when it again came down to normal. The patient never lost her toxic appearance even after the operation. Blood cultures were negative, Widal negative twice, malaria negative. The patient never had a chill, but did have sweats with her temperature rises. Neurological examination was negative. A very reliable medical man made a diagnosis of pneumonia, and an X-ray of the chest confirmed the presence of some pathology in the lungs, but not typical pneumonia. The high temperature kept up for about a week, when it came down to normal and it was supposed that this was a pneumonic crisis. But after this normal temperature when the fever made two more excursions in two days I felt that I had no right to delay any longer and determined to again investigate the sinus. I exposed the sinus once more and the only abnormal appearance was a slight pallor. I had, however, made up my mind to investigate it thoroughly and ligate the jugular vein, whatever condition might be present. This I did and when I opened the sinus I found a dram of pus in it. It seems to me that the sinus inflammation was very likely present when I first suspected it and exposed it and that the pneumonic condition was secondary to the sinus infection. The only thing I had to go by was the presence of an otogenous etiology, the non-amelioration of symptoms after the mastoid operation and the exclusion of other ailments, excepting the pneumonic condition. Were it not for the pathology in the lung I might have decided to open the sinus sooner. I might also state that the gradually declining hemoglobin picture induced me to concentrate upon the sinus. This point

is excellently brought out by Kopetzky, and has been worked out in a number of mastoid cases by Ersner. The particular point this blood picture brings out is that there is a gradual destruction of the hemoglobin element in the blood when the streptococcus hemolyticus is the offending organism, and as is well known that this organism is the cause of most cases of sinus thrombosis, its activity in destroying the hemoglobin may be utilized as an index of blood stream infection. The lesson in this case would seem to point to the fact that we must be extremely careful, where everything is atypical, to make definitely sure of the other complicating conditions as to their etiology and diagnosis before the sinus is eliminated from blame; but also be sure that the medical condition does not explain all of the patient's illness before you attach the guilt to the sinus. And where there is any question at all about the certainty of the complicating condition proceed to rapidly eliminate the sinus by operative means.

There is one phase of sinus thrombosis which one should bear in mind, particularly when dealing with young children and infants, and that is primary jugular bulb thrombosis. Here, probably because of some dehiscence in the floor of the middle ear, the bulb of the sinus is directly infected from an otitis media without even mastoid involvement. It is most difficult to make up one's mind as to its presence, but when one has this condition in mind it goes a long way towards the diagnosis. McKernon's suggestion is helpful. He thinks that when an infant who is suffering from an acute otitis media and suddenly develops a high temperature, which rapidly comes down to normal with the patient again in apparently good condition, but suddenly a day or so later the same temperature phenomenon repeats itself, the patient is probably suffering from a primary jugular bulb thrombosis. There is usually no mastoid involvement, but a high leucocytosis of about 20,000, with a high polymorphonuclear count. Blood culture if positive is helpful. When we recall, however, that children so easily run up a high temperature and so frequently have otitis media on both sides and that symptoms of many of their complaints are masked for a considerable time, we realize that it is not an easy responsibility to make a decision to explore the sinus. If the otitis media should be bilateral, the Ayer-Toby test may be employed, but I certainly would not depend a great deal on it. There remains only one or two things to do: First, to think of the possibility of this condition occurring and, secondly, to be extremely cautious and permit ample time to rule out other medical or surgical conditions, make blood cultures and frequent blood counts, having the constantly diminishing hemoglobin picture in mind (as pointed out by Kopetzky) and when things do not look

very promising one is justified in investigating the lateral sinuses. It is undoubtedly very important to make an early diagnosis, but the operative procedures involved are too serious to be undertaken without a great deal of deliberation.

Another type of sinus thrombosis one may have to contend with is one of cavernous sinus thrombosis. While it is more commonly caused by infections of the face and nasal sinuses, occasionally it may be caused by an extension from a lateral sinus infection. The chief diagnostic sign, beside symptoms of septic infection, is swelling of the tissues of the orbit. There was one case in our service, a patient of Dr. Ersner's, who also complained of severe pain behind the eye, which was persistent and was present before the orbital edema appeared. This patient had a lateral sinus thrombosis and an attempt to drain the cavernous sinus through the superior petrosal sinus was made but was futile. The prognosis in these cases is always bad. Eagleton claims to have cured some by ligating the internal carotid artery. Mosher advocated drainage after enucleation of the eye on the affected side. If the diagnosis is positive anything that can be done seems justifiable in the present state of our helplessness with this condition. When, however, the diagnosis is uncertain we cannot afford to be so bold.

I wish, here, to recall a case of mine of acute mastoiditis in a young child which scared me considerably. The child was running a rather high temperature after the operation and one day I came in and found a typically edematous eye on the side of the mastoid involved. I was greatly relieved by finding an abscess of the scalp, which accounted for the edema. This is another example of how a simple thing may appear like an insurmountable mountain.

The symptoms of vomiting in the course of an acute or chronic otitis media may have important significance and deserves careful consideration, yet ordinarily while it may prey on our minds it is of very little help in reaching a conclusion as to its meaning. It may occur as a result of so many things, like digestive disturbances, fever, etc., that if it had an important cranial etiology it could easily be overlooked. In a patient, however, who is in the postoperative period of mastoidectomy, particularly in a chronic condition, it is an alarming symptom. It may mean the initial or meningitic stage of brain abscess, cerebral or cerebellar. It may mean the beginning of a generalized meningitis. It may mean a labyrinthitis. Which of those it may be depends upon the combination of symptoms with which it is associated. Macewen states that in cerebral abscess it is only present in the initial stage and with this many otologists agree, but it is rarely a prominent symptom in this condition. In cerebellar

abscess it is not only present early, but may recur frequently and become one of the cardinal symptoms. It was the first symptom to attract my attention to the cerebellum in my case of cerebellar abscess. If it is associated with vertigo we focus our attention on two conditions, namely, cerebellar abscess and labyrinthitis. If it is projectile in character we think of it as intracranial. If it is accompanied by nausea we are more likely to think of the labyrinth. Kerrison's statement regarding this symptom is interesting and helpful. He states that recurrent vomiting, accompanied by vertigo, rotary nystagmus and nausea, the symptoms showing signs of abatement, is characteristic of labyrinthitis. If no nausea is present and the symptoms tend to get worse it is probably a cerebellar abscess.

Let us focus our attention for a few moments on the labyrinth. Diffuse suppurative labyrinthitis is always accompanied by vertigo. Vertigo is a symptom which is more prominent when the patient is up and about than when in bed. So this symptom in a postoperative patient must be sought. It can often be recognized by the position the patient occupies in bed, in which he will not move in a certain direction for fear of getting dizzy. He lies on the side toward which is directed the quick component of the accompanying nystagmus—that is, on the sound side—so that he may turn his eyes up when spoken to, or to look around, in the direction of his slow component, which diminishes his vertigo. Such a picture is definitely one of labyrinthitis. The nystagmus is always present and rotary in character. The treatment is watchful waiting, with a keen eye directed to developing signs of meningitis, when operation is immediately indicated. There is one phase which requires mention and that is that this train of symptoms may appear in a postoperative case as a result of hyperemia of the labyrinth, which is caused by inflammatory congestion in adjacent structures. It particularly deserves mention because one of the accepted indications for a labyrinthectomy is developing signs of labyrinthitis following a radical mastoid operation where there may be some suspicion of a probable injury to the stapes during operation. I saw two such cases, one of Dr. Ersner's and one of my own, in which these symptoms developed after a radical mastoidectomy and which subsided in Ersner's case in about three days and in my case in about twenty-four hours. Had we been in a hurry to follow this dictum, both of our patients might have had a needless labyrinthectomy done. The indications for a labyrinth operation are more or less definitely accepted and there is never any urgency for immediate operation, with the probable exception of developing signs of meningeal involvement.

There are two more intracranial conditions complicating middle ear disease which rank foremost in seriousness and probably second to sinus thrombosis in frequency of occurrence, and these are meningitis and brain abscess, either cerebral or cerebellar. Both of these conditions attract our attention first and foremost by their headache and, secondly, by the change in the individual's mentality. In brain abscess the meningeal involvement being of short duration, the headache is less prominent and often fades out of the picture, while the change in mentality remains. With meningitis, if it tends to become generalized, everything about it is and remains in a conspicuous foreground. This is true of the headache, the fever, vomiting and the mental change. There is nothing characteristic about the fever except that it is high and constant. The headache is severe and the vomiting persistent. The mentality throughout the disease is one of activity, irritability and alertness till the end, when coma supervenes. The quickened mentality will occasionally be helpful as a differential sign between meningitis and brain abscess when the latter condition is suspected. I recall one case which was sent in to the hospital as a suspicious case of brain abscess, but upon seeing the patient I made up my mind that the condition was more like a meningitis than a brain abscess because of the patient's alertness and quickness of mind. He asked too many questions about himself, grasped the answers quickly and asked another question almost before one was answered. Lumbar puncture proved this case to be one of pneumococcic meningitis.

The diagnosis of a frank case of meningitis is not hard to make. The few prominent symptoms just mentioned, together with rigidity of the neck, photophobia, Kernig's and Brudzinski's signs, ankle clonus and tache cerebrale, are nearly always present. Various cranial nerves may be involved at the base of the brain, giving different palsies. A lumbar puncture, of course, settles the diagnosis, not only of meningitis, but also of the type.

The fear to do a lumbar puncture where another condition in the head is suspected, like brain abscess or sinus thrombosis, is probably founded on some untoward experiences, but in spite of that, one is not justified in remaining in the dark because of what happened in isolated cases. Toby claims to have done hundreds of punctures in patients, many of whom had sinus thrombosis, without any ill effects. A careful lumbar puncture, withdrawing a small amount of fluid, should be done where meningitis is suspected and in all intracranial conditions for that matter. The type of meningitis present is of practically little importance in so far as treatment is concerned, except in epidemic cerebrospinal meningitis, where specific treatment

is indicated. In the others it is of prognostic importance; where a cloudy fluid is found but no organism (protective meningitis, Eagleton), the prognosis is more favorable.

The condition spoken of as localized meningitis and viewed as a distinct entity, without accounting for its focus of origin and mode of developement, is to my mind not only overplayed but is a distinct handicap and harmful to proper diagnosis in certain instances. We have one definite so-called entity of localized meningitis in otology, known as the Gradenigo syndrome, where we have an abducens paralysis with fifth nerve neuralgia, but then here, too, we have the ear as a point of origin and an anatomic explanation for its mode of development has been demonstrated. I have seen one case diagnosed as localized meningitis, of unknown origin, in the region of the optic chiasm, with the added provision that it might have been due to syphilis, the condition which bears the blame for almost everything we don't know. It was so diagnosed because of some visual field studies. The patient got well and later a sphenoiditis proved to be the cause, which was not apparent at first. The reason I mention this subject is because I have seen neurologists make that diagnosis in one of Dr. Ersner's cases of temporosphenoidal abscess in which there was a third nerve palsy and they insisted that it was a meningitis at the base and delayed operative measures for a considerable time, because they would not see it in the light of brain abscess, as it occurred to me. If I recall correctly, they even refused to make a lumbar puncture because they feared that the meningitis was walled off and they would not risk generalizing the infection.

We will now turn to the subject of brain abscess. In this we will include cerebral as well as cerebellar abscess as they are alike in many respects, the chief difference being in the localizing signs, which can be pointed out as we go along. The subject of brain abscess is probably the most important of the cranial conditions with which we must deal, for the following reasons: First, because the symptoms are most obscure and often very few in number; secondly, abscesses are nearly always fatal unless relieved by surgical means, and thirdly, there is no other condition where needless surgery can do so much damage, and yet the earlier surgery is undertaken the better are the chances for recovery. In a mistaken sinus thrombosis we may open the lateral sinus, even ligate the jugular vein, and not expect, as a rule, trouble resulting directly from the surgical procedure. In meningitis, with fairly conservative management we cannot do much harm in cases which will get well, and in those cases which will not get well you cannot do more harm. In brain abscess, however, there is always the question of correct localization and if

searched for in a region where it is not present, this nearly always being done through an infected field, the danger of creating an abscess is apparent.

The symptoms which make us think of brain abscess are very few in number and when they arise careful and constant neurological observation must be made to detect the earliest possible evidence of organic involvement. The first and foremost symptom is the change of the patients' mental attitude into one of depression, with a markedly retarded cerebration. This may manifest itself in failing memory, listlessness and inability to sustain attention on any subject. The patient seems to live as if in a dream. He is asked a question and he will look at you for a while, then seem to forget about it—but will answer it a little later. When this appears in a patient with a chronic otorrhea or in the postoperative period of a mastoidectomy we should at once suspect an abscess. When there is a history of recent annoying headache, with possibly rigors or chilly sensations, with some fever, our suspicion is considerably enhanced. When the patient appears to have lost considerable weight in a short space of time it further adds to the picture. Vomiting, added to this group of symptoms, adds another strong indication. When these are present, the vomiting persistent, the headache more or less constant, with some evidence of vertigo, the cerebellum should be strongly suspected. There may be a little elevation in temperature occasionally, or the pulse, temperature and respirations may be normal. When we have the typical picture of subnormal temperature, pulse and respirations we know that there is intracranial pressure, but this picture is rarely seen and almost never in the early stages. There is one feature in the temperature which should always be looked for and that is an occasional falling of the pulse to subnormal in the presence of a normal temperature, or even some elevation in temperature. This sign, which was pointed out by Macewen, has never failed us when it was present and it was present often. It was the first sign that gave us the clue in one case when everything seemed to point to basal meningitis. It was present in one of my cases of cerebral abscess, even in the presence of a concomitant sinus thrombosis and subdural abscess in the parietal region, which was in effect an extensive meningitis in that area.

The above picture may be present, perhaps, in its entirety. That is one thing, but to open the skull and explore the brain is another—we must know where to search and, besides, many of the symptoms may be the result of some other causative factor.

From here on we must depend upon our neurological knowledge and neurological signs. I wish I could say, instead, the neurologist

and thus bring him in to share the responsibility. He should really be the logical man to find our abscess for us. Our experience in this respect, however, has not always been completely satisfactory.

If I may digress for a moment I would like to say that here is a responsibility which the neurologist should fully realize. His head, however, is too full of other neurology and psychiatry to pay much attention to brain abscess from the standpoint of the ear. As a rule when he is asked to examine a patient he looks at him from a purely neurological standpoint and tries to explain the findings on the smallest area involvement which could produce a certain combination of signs. For example, in one case the presence of a partial motor oculi paresis and abducens paralysis spelled basal meningitis, while in reality these occurred because of constantly increasing pressure exerted by a constantly increasing brain abscess. In short, the neurologist did not take into account the clinical course as it started with the extending pathology from the ear. My contention is that it is the duty of the neurologist to study sufficient otology to be able to give us the advantage of his detailed knowledge of the brain viewed from the ear and not alone from the brain. Some of our good text-books on neurology, in discussing brain abscess tell us that ear conditions are the most frequent cause of brain abscess, and yet go through the formality of stating the usual divisions of brain abscess into the various stages and not a word connecting the symptomatology with the pathology of the ear. I have heard a neurologist report a certain case in which a cause was searched for which would explain certain symptoms which as they appeared to me could have formed part of the picture of otitic brain abscess and yet not a word was mentioned about the condition or examination of the ears. In fact, when I specifically asked a question I was told that as far as is known the patient had never complained about his ears.

However, we should not shift all the responsibility upon the neurologist. Otologists should have some idea of neurology so that they may be able to follow the pathology from the ear into the brain.

To be able to do this a few neurological facts concerning the locations in which otitic brain abscesses may form should be known.

First of all, we must visualize what a brain abscess does when it forms in the brain; for instance, that it gradually increases in size, not so much by displacement of tissue as by destruction and replacement of broken down material in its formation, and exerts pressure in all directions, thus affecting the neighboring structures mildly at first, then in a more pronounced fashion. There is always an accompanying encephalitis which entails pressure from a surrounding

edema and which may shift from one position to another and thus certain signs may disappear for a while.

It is a well established fact that the great majority of otitic abscessses occur in the temporosphenoidal lobe and cerebellum, the former having the greater number.

The nature of some of the more important surrounding structures should be known, as they are sooner or later impinged upon by the pathological process.

To search for an abscess which is as large as a pea or thereabouts, one must be a superhuman sharpshooter to locate it with a searching ing instrument, so the average man will very likely have to search for a more or less larger abscess.

The important structures in the temporosphenoidal lobe are the word hearing centers on its outer posterior aspect, so that if an abscess were on its cortex in that region, and this is quite close to the ear, the inability to name familiar objects would be manifest. An abscess is more likely to be situated in the white matter of this lobe or, as it is termed, subcortical abscess, so we must have some idea of the fibrous structures in that region, and know that these fibres may be destroyed or involved by the surrounding encephalitis and their function temporarily or partially impaired. In speaking of fibres we must remember that these may be projectional fibres or those which leave the brain into the spinal cord, and commissural fibres or those which connect one side of the brain with the other, and association fibres or those which connect one part of the brain with another on the same side. For instance, you give a man a pencil and paper and tell him to write the word pencil; he sees the pencil, recognizes the word and its meaning, goes through the complex motion, which indicates that he knows what to do with the pencil and paper, and writes the word correctly. These processes represent a fibrous connection between the word hearing center, the visual center, the center for carrying out purposeful motion, and the writing center, as well as the motor center. If the individual should fail in any part of this procedure, then either the center or the fibres connecting the centers are at fault. Any one of these actions may be at fault in a temporosphenoidal abscess, but the most common is the word hearing center. If the patient does not recognize a word, but recalls its uses when reminded, he has a partial naming aphasia, this being the most common of all types of aphasia in this type of abscess. If he does not even recall a thing about the word when it is mentioned to him, he has a complete naming aphasia. The word hearing center at the posterior part of the second temporal convolution is at fault, and most likely at the cortex.

To follow our abscess—if it should press upwards it would gradually involve the subcortical motor fibres, which come from the cortex in the following order: First, those which go to the face on the opposite side, then the upper extremity, and then the lower extremity. So that when first fibrillations or mild spasmodic contractions with some spasticity, then weakness, and finally paralysis sets in in the order mentioned it is almost certain that there is a temporosphenoidal abscess present. If the patient is seen in the earlier part of this picture one can at least say that its presence is highly probable.

Passing inward and downward, structures at the base of the brain in that region would be involved. The third nerve is the earliest structure, then the fourth and sixth nerves would be involved. All of these nerves supply the extraocular muscles.

An encephalitis or an abscess pressing inward or an abscess located deeply inward would involve the internal capsule and cause sudden paralysis of the extremities of the opposite side. I have seen one case with an abscess in the internal capsule which, because of its suddenness in appearance, was at first mistaken for an apoplexy, but the neurologist diagnosed an abscess, which was found at operation. Dr. Coates had one case with widespread paralysis, due to multiple abscesses destroying the fibres as they came from the various centers in the cortex.

An encephalitis in the temporosphenoidal lobe could involve the fibres of the optic radiation and cause disturbances in the visual field —an homonymous hemianoptia—or may only partially involve the fibres. Eagleton has taken advantage of these visual field phenomena and discovered that very early in temporosphenoidal lobe abscess, because of the edema due to an encephalitis, there are indentations in the field of vision, these indentations disappearing at the next examination and reappearing at subsequent examinations. It is here that the ophthalmologist could be of the greatest assistance to the otologist if he would take daily visual field readings. This would be of greater help than eye ground examinations, which are of value only when there is positive involvement of the optic disc at a late stage of the disease. In interpreting these visual fields one must be careful to note the disappearance and reappearance of these indentations as more permanent ones could be due to other causes, like retinal or optic nerve disease. We must also not forget that the visual field reading depends greatly upon the patient's answers and their value is markedly diminished in a patient who is not overly intelligent.

If the cerebellum is involved the pointing tests or other co-ordination tests would be of considerable help if present. Thus the patient may past point with the finger on the side involved. He may have

difficulty in locating the tip of his nose with his finger or have difficulty in locating parts of his lower limb with the lower limb of the side involved; for example, place his heels to the knee or toe. Adiokokinesis may be present, as could be tested by the inability to rotate both wrists at the same rate of speed, the side involved lagging behind. There may be hypermetropia or overpointing when directly attempting to point to an object.

The facial nerve may be involved by pressure, causing facial weakness on the same side of the lesion. Other pontile nuclei or nuclei in the medulla may be involved by pressure. Facial neuralgias may be present from pressure on fifth nerve. There may be protrusion of the tongue to the diseased side because of the weakness of the muscles on this side fail to protrude its half of the tongue.

Let me now for the sake of clearness rapidly summarize the things one would look for in a case of suspected otitic brain abscess at the bedside. We would attempt to rule out meningitis by testing for rigidity of the neck, Kernig sign, Brudzinski sign, tache cerebrale, Babinski sign and clonus.

We would then try to rule out cranial nerve involvement. The sense of smell may be tested on each side, but this is of no great importance. Have the eye man, if you cannot yourself interpret eye grounds, examine them, as well as make visual field studies. Have patient rotate the eyes in all directions, following your finger. If there is weakness, ptosis, one dilated pupil, or perhaps some squint and diplopia is complained of, your mind's eye is carried to the cerebral peduncle, where the third nerve emerges and is probably involved by pressure from a neighboring temporosphenoidal abscess. If there is an internal squint accompanied by facial pain, it is probably the Gradenigo syndrome. Look for paresis of the facial muscles, particularly on the opposite side in cerebral, and on the same side in cerebellar abscess. In the latter instance the forehead is probably also involved, while in cerebral abscess it is not likely to be affected. Determine if voice or swallowing is affected. Look for deviation of the tongue to determine hypoglossal nerve involvement. Taste may be tested in the anterior and posterior parts of the tongue. Then see if you can notice any fibrillations in the face, upper extremity and lower extremity, or determine if there is a history of such twitchings. Search for weakness or paralyses of these muscles. Show the patient a familiar object and ask him to name it to determine any naming aphasia. Have him write his name or other word and thus note if there is any visual, motor or writing center affection. Note any change in speech. Give him an object like a pen-

knife and ask him to use it and see if he carries out these purposeful motions correctly.

Test his pointing or co-ordination movements, like rotating his wrists, point finger to nose, or heel to opposite toes. Test his falling to see if he falls in the same direction at all times, independent of the position of the head.

If you rapidly go through these tests and you find them negative, then there are two probabilities; first, that there is no abscess or, secondly, if there is one you have not succeeded in localizing it. To search for it on any one, or even what may appear the most likely side, will probably be disappointing. In fact, you may at postmortem find that the abscess is at a distance from where you searched and just as likely the opposite side. If, however, there are sufficient signs present to point to the region interrogated by these tests, even when they are mild, you are justified in making an exploration.

Some Details in Diagnosis. By this I refer to various tests or so-called signs of such and such a disease, and also to empirical statements made by various authors who have found them to be of value. Of course, no positive diagnosis can be made on any of those signs. Very often they are hard to elicit and when we think we have found them we would very likely hesitate to undertake any surgery unless they are accompanied by more reliable data.

The first I wish to refer to is the Ayer-Toby test in sinus thrombosis. In this test we note the spinal fluid pressure with a manometer and while taking this record the internal jugular vein in the neck opposite to the suspected side is compressed. This is supposed to raise the intracranial pressure by blocking the venous return from the head. If there is an elevation of about 10 m.m. or more in the spinal pressure it indicates a thrombus or block in the suspected side. For, if it were not blocked it would permit a good return of blood through the noncompressed side and the pressure would not be raised.

Our experience with this test in several cases had not been helpful. In one case of mine I did the test about a week before the sinus operation. During the operation I found pus in the sinus, which would make one believe that at the time the test was made there must have been a thrombus or complete block of the vein, yet the test was entirely negative.

Griesingers' Tests: One test consists in compressing the supposed normal vein and if the other vein is blocked there should be a flushing of the face and a fullness of the retinal vessels. One could easily see that in this test the personal element of the observer would be a great factor. What constitutes a slightly fuller than normal

vessel in the retina or a barely perceptible flush of the face are very uncertain data to depend upon when the question of operation is still open.

Another of Griesinger's signs is one in which there is a lower postauricular swelling without pus formation. This is supposed to indicate a blocking of the vessels, therefore producing edema. I saw what I thought was this sign in one case of sinus thrombosis, but this occurred after the mastoid operation. There was considerable tumefaction in the postauricular region which did not yield pus on probing. I looked upon it as an inflammatory reaction and I certainly would hesitate to undertake surgery on its strength.

However, there is value in each one of those signs and we should look for them, and sometimes, in the presence of a certain amount of unbalanced data, they may be the deciding factor.

Yerger has made some studies on the cell count of the cerebrospinal fluid and reported some interesting data which may be of value in helping to establish the diagnosis of some intracranial infection. He found the cell count increased in all intracranial inflammations. In extradural abscess and in sinus thrombosis the cell count ranges between 25 and 250. In subdural abscess and brain abscess the range is between 250 and 2500. Anything above that has proven to be meningitis. In any event, the cell count shows the presence of some infective irritant within the skull and should be made in all suspicious cases.

Gowers' Syndrome: A persistent headache with persistent vomiting points to intracranial complications. This combination when it occurs in a young child, Eagleton thinks, indicates almost certainly a brain abscess.

In brain abscess there is always a restlessness and insomnia, yet the patient is depressed and sleepy, so that when he tries to sleep he is unable to do so. This peculiar situation was described by Kerrison.

Yawning and slow speech have been found to be frequent symptoms in cerebellar abscess.

Dandy attempts to locate the shadow of an abscess by radiography after injecting the lateral ventricles with air. This is quite a formidable undertaking for diagnostic purpose, particularly when there is only a mere suspicion of the presenec of an abscess.

Convulsions: It has been Eagleton's experience that a sudden onset of convulsion in the presence of a possible focus of infection has proven to be cerebral abscess. This particularly being true in children. Our cases which showed convulsions usually did so in the later and terminal stages.

The Barany tests have been successfully used by McCuen Smith and Fisher in finding a cerebral abscess when there were responses from canals of one plane and none in the canals in the other plane. Spontaneous vertical nystagmus has been observed by them in brain abscess. We have tried these tests in some of our cases which proved to be cerebral abscess and obtained negative findings. In cerebellar conditions these tests have been of more definite value.

We will not devote a few words to the history of the patient under suspicion of intracranial disease. This, almost at the close of this paper, seems a rather belated time to speak of history, but I do this not without a reason. When we read the history on the average hospital chart we find it replete with data which have no bearing on the condition at hand. The resident who usually takes the history could not be expected to be sufficiently versed in intracranial disease to be able to search for just the thing which may throw light on the possible development of intracranial lesions. It is therefore important for the surgeon to take his own history in these cases and attempt to extract information which the patient himself does not give or has forgotten. The duration of his otorrhea seems unimportant to him. The fact that one time recently he had a little headache and a chill with some discomfort about the ear passes off as a little grippe which is promptly forgotten. These facts must be searched for because they are not as a rule voluntarily offered. The same may be said about making certain observations of the patient while in the hospital. The nurse, or even the resident, may not know that it is important to make frequent observations as to the presence of fibrillations in certain muscles or slight weakness of the face or limbs, or if they did notice it, the time and order of its appearance may not have been thought important enough to have been noted. A nurse will tell you that the patient vomited, but will never know whether the patient had had slight nausea unless she is specifically asked to look for it. Vertigo while the patient is in bed may never be known unless the patient is asked about it in various ways. My case of cerebellar abscess never complained of vertigo and no one ever knew until after many questions the patient told me that at one time she thought that things in the room stood upside down, but she did not say anything to anyone. One cannot be present to make all observations day and night, but if word is left to look for certain symptoms the nurse will usually detect them at the earliest possible moment.

Treatment: It is not the purpose of this paper to describe any treatment which is more accurately described in textbooks. There are only a few points which seem worthy of mention.

First of all, I wish to emphasize the dangers of a sclerotic mastoid. This type is usually looked upon as a healed inflammation, but in every chronic case of ours that had intracranial complications we found a hard sclerosed mastoid on the surface and necrosis and infection at the depth oft he bone. It would seem to me that, given a chronic otorrhea which is followed by mastoid pain and the X-rays show a sclerosis of the mastoid, it is most dangerous to wait till the condition subsides. Nothing short of a radical mastoidectomy is compatible with the safety of the patient.

In sinus thrombosis operations the packs which control the sinus bleeding are usually left in for several days. In one case of mine, which had also two brain abscesses, I found at postmortem examination that the torcular herophili was filled with pus. The infection here probably extended backwards behind the pack, not having any exit anteriorily. I could not in this case blame the death of the patient to this condition as there were more serious conditions present which caused the fatality. I carried this in mind, however, till my next case. Here at the first dressing within twenty-four hours I decided to remove these packs. I was ready to pack again should there be free bleeding, but to my delightful surprise there was none. It would seem that beside exposing the sinus far beyond the probable area of infection it is well to remove the packing as early as possible to avoid backward infection.

In meningitis the conservative treatment is to eradicate the focus of infection and perhaps make some small incisions in the dura for the purpose of drainage. There have been various elaborate operations devised for the surgical treatment of meningitis, *e. g.*, Eagleton's subarachnoid irrigation, Kopetzky's trephine of the cisterna magna, etc., but the pendulum seems to have swung the other way again to the conservative treatment. It stands to reason that had these methods real value more cures should have been reported than have been. It seems to me that the markedly diminished mortality in epidemic cerebrospinal meningitis by a specific serum points toward the right direction. The greatest hope probably lies in the discovery by the laboratory of a specific serum or chemotherapeutic agent which will be used in conjunction with the rational surgical procedure of eradication of the focus of infection.

In brain abscess recoveries reported were in the majority of cases in which drainage was instituted early in the most conservative way. King's method of treatment is the most radical departure. He uncaps the abscess cavity, herniates it and keeps it clean with Dakin's solution. Where the structures can be spared, this method, because

of King's excellent results, deserves further trial. Usually it is hard to judge which structures of the brain can be removed without seriour damage. I had one experience with this method not of my own choice. I searched for an abscess by making a small incision in the dura, this followed by immediate herniation of the brain tissue, which was necrotic. There was nothing left for me to do but to enlarge the dural opening to prevent strangulation and remove all the necrotic brain tissue. This patient also had a purulent meningitis, sinus thrombosis, and died, so the effects of this method of treatment cannot in this case be judged fairly. The patient, however, had a naming aphasia after the operation, but the herniated brain seemed to have receded fairly well in the ten days that the patient lived following the operation.

My case of cerebellar abscess got well merely by puncturing and inserting a small gauze drain into the cavity and changing it daily. This favors the more conservative form of treatment.

It can be said about brain abscess that when localization will be more exact and in a very early stage, the prognosis will be quite favorable and I also believe that this will be true with the most conservative forms of drainage. We never know just what the functions of certain brain tissues are because at present they seem to us to be silent areas.

In conclusion, I wish to express my grateful appreciation to my chief, Dr. Coates, for the opportunities offered in managing these cases, as well as for the most helpful advice, of which he freely gave. His excellent judgment and kindly disposotion have been, during the most critical period in these cases, of a greater comfort than words could express. I cannot help but to express my feelings that to work under the instruction and guidance of a chief such as he is, it not only an unbounded pleasure but the rarest of opportunities.

1717 Pine Street.

A CASE OF CHOANAL ATRESIA.

Dr. Paul L. Mahoney, Little Rock, Ark.

A resume of the literature shows that only 180 cases of choanal atresia have been reported to date, and I wish to add the following case to this number.

I was called in consultation with a pediatrician to see a greatly emaciated male child, age 3 months. The history was that since birth this child had eaten and breathed with difficulty and has had a thick mucus discharge from both nares. He also had suffered numerous respiratory and gastrointestinal upsets.

Examination: Throat shows no pathology of interest. There is a thick mucus secretion from both nares, and breathing is difficult. After cleaning the nares the membrane covering the turbinates and septum appears waterlogged. Attempts to force colored solution through nares into postnasal space were unsuccessful. The passage of a probe met with failure, striking an obstruction. The hard palate was arched only slightly. The eardrums were slightly retracted.

Operation: Under general anesthesia openings were made through the occluded choana, which appeared to be of membrano-osseous consistence. These openings were enlarged as much as possible. I passed a large rubber tube through one naris, brought it through the opposite side and tied at the columetta. The after-treatment consisted of cleansing only, no sounding. This tube was removed after two weeks.

At this date, nine months after operation, the child has attained apparently normal weight and health. The nasal mucous membrane has attained a normal appearance, all symptoms have subsided, and I feel that the openings made are permanent.

831 New Donaghey Building.

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THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Detroit on Sept. 12, during the session of the American Academy of Ophthalmology and Otolaryngology; 102 applicants appeared for examinations, with .107 per cent failures.

An examination was held in Memphis on Nov. 14, preceding the session of the Southern Medical Association, with .127 per cent failures.

In the course of the past year, 369 applicants have been examined.

In 1928, examinations will be held in Minneapolis, on June 11, at the session of the American Medical Association, and in St. Louis, on Oct. 15, during the meeting of the American Academy of Ophthalmology and Otolaryngology.

Prospective applicants for certificates should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha, for proper application blanks.

Dr. W. P. Wherry, *Secretary.* Dr. H. P. Mosher, *President.*

BARBITAL TO PREVENT TOXICOSIS FROM LOCAL ANESTHETICS.*

DR. C. B. WILLIAMS, Mineral Wells, Tex.

In the routine use of local anesthetics for operative work there are two things which for some years have given me both concern and annoyance—concern from the frank toxicosis which we now and then meet, and annoyance from those psychic manifestations which often simulate toxicosis and which at least always result in delay and in anxiety on the part of friends or relatives of the patient who may be present.

I have for years tried to forestall this concern and annoyance by the administration of morphin and atropin prior to beginning the use of local anesthetics, but have been so often disappointed that of recent years I have come to feel less secure in this method.

Tatum, Atkinson and Collins[1] reported in 1925 from the laboratory of pharmacology of the University of Chicago that experimental work which they had done on dogs demonstrated that the preliminary intravenous use of sodium barbital and paraldehyde would increase the tolerance to cocain some 400 per cent.

Notwithstanding the fact that this report attracted my attention at the time, I did not become interested in the subject till Leshure[2] gave his favorable conclusions in a preliminary report on the clinical use of barbital or its sodium salt by mouth in 100 cases. He said, in part, that "for a number of years I have administered to all patients requiring operation under local anesthesia morphin either alone or in combination with atropin or scopolamin. In spite of this precaution, a considerable number of these patients have exhibited symptoms of poisoning, such as pale, cold, clammy skin, rapid and feeble heart action, and convulsive tremors.

"During the last year I have substituted for these drugs diethyl barbituric acid (barbital) or its sodium salt (sodium barbital), with the result that none of the 100 patients so treated have manifested any of the symptoms mentioned above, and the natural inference is that barbital is an efficient antidote to cocain, procain hydrochlorid and other similar drugs used to induce local anesthesia."

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*Read before the Northwest Texas District Medical Association, Wichita Falls, September 14, 1927.

During the last 90 days I have substituted barbital for morphin and atropin in 52 cases, random — not selected — including tonsillectomies, adenectomies, submucous resections of the nasal septum, antrotomies, etc., etc. In only two instances were there any symptoms suggestive of toxicosis and these were transient, causing a delay of only two or three minutes.

I have noted particularly that while most of these patients were noticeably nervous when they came to the office to arrange for operation, they would return at the appointed time quite calm, and their cooperation throughout was, on the whole, rather to be remarked upon.

It would thus appear from these few cases that from a clinical standpoint barbital is a more efficacious antidote for toxicosis in local anesthetics than morphin and atropin, that its administration prior to the use of local anesthesia for operative purposes has a distinctive value and that co-operation of the patient seems more certainly assured.

METHOD OF ADMINISTRATION.

I have the patient procure two 5-gr. tablets of barbital with directions that one tablet be taken by mouth with a glass of water two hours before the appointed time for operation, and the other one hour before.

I have felt that in certain of these patients it would have been better to give 15 grains in two doses, but as a means of getting a test with a definite dosage I have adhered to the administration of 10 grains in each of these 50 cases.

Comment: The question might well be raised that in so few cases there might have been none who would have shown any untoward symptoms even though no antidote had been administered. To this I would reply that, for the present at least, I have that vague but positive conviction of the superiority of barbital in such cases which has caused me to adopt it as routine instead of morphin and atropin.

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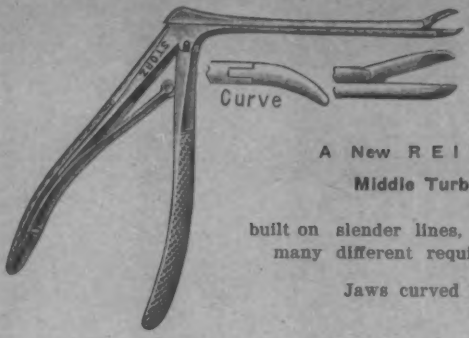

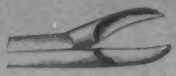

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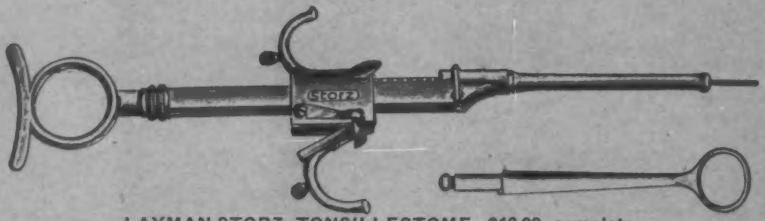
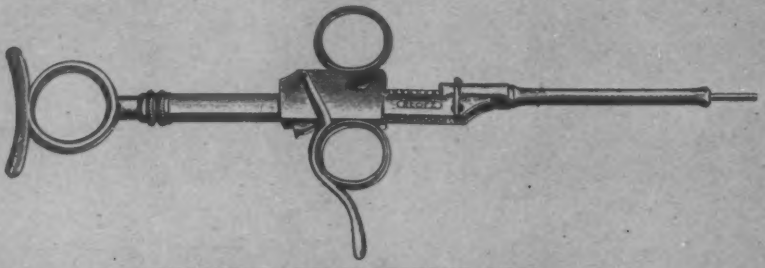


LAYMAN-STORZ TONSILLECTOME, $12.00 complete.

The self-locking snare. Clutch opens and closes automatically. Simple to operate.

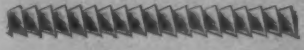


BECK-SCHENCK TONSILLECTOME, $12.00 complete.

Both models are equipped with our new type of threadbar, and are furnished with a straight canula and wire carrier, a medium size Vedder tip and 1 dozen twisted wire loops. Vedder tips can be furnished in 3 sizes. Extra wire loops, 50c per doz.

Old Type

New Type



Our new type of thread, which is much more rigid than the old, has spiral ridges with a square shoulder on the side where they meet the clutch when pressure is applied, in contrast to the old type, which tends to slip off on account of being on a slant.

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